

A STUDY ON KALANCHAGA PADAI

(DISSERTATION SUBJECT)



*for the partial fulfillment of the requirements
to the degree of*

DOCTOR OF MEDICINE (SIDDHA)

Branch III – SIRAPPU MARUTHUVAM

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INTRODUCTION

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Siddha system of medicine is a tradition one with prestigious background of Tamil culture. It's perhaps the earliest medical science that laid stress on positive health , a harmonious blending of physical , mental, social ,moral and spiritual welfare of an individual .

The siddha system of medicine is believed to be originated from “ **lord shiva** ,” The supreme God of Tamil and he is considered to be the child of “**Siddhars** “Lord shiva preached this science to “Shakthi “ the Goddess and then to “nandhi from then to common people by siddhars .

Siddhars in their attempt to elevate themselves to a perfected earthly immortal [attain siddhi] developed techniques which included controlled breathing [vaasi], concentration of mind, intense meditation besides dietary regimen and certain postures for psychosomatic harmony.

The paramount aim and object of siddhars is to attain enlightenment for which they needed a strong body, sound mind and longer life span on pursuit of such endeavors they developed a science, yoga, rejuvenation techniques , alchemy, varma, etc...

“Health is wealth “

It is well known for all. We all accept this for without good health nothing is possible. The success one attains by means of physical or mental power must be only through good health. When there is harm to health everything becomes stagnant. Hence it is

very essential to maintain good health . However,when complexities arise , there must be a remedy. During such situation, the “siddha system of medicine “ come to rescue to cure .

The universe consists of two essential entities .

1. Matter
2. Energy

Matter cannot exist without energy and energy cannot exist without matter , the two exist are inseparable.

In siddha system of medicine the understanding of human body mechanism starts from the knowledge of cosmogenesis. The nature and human being are interrelated.

“mz;l;jpYs;sNj gpz;lk;
gpz;l;jpYs;sNj mz;lk;
mz;lKk; gpz;lKk; xd;Nw
mwpe;Jjhd; ghh;f;Fk; NghNj”

-rl;lKdp Qhdk;.

Man is said to be the microcosm and universe is macrocosm. What exists in the universe exists in man. The universe made up of 5 basic elements “**Pancha boothams** “

[gQ;rG+jq;fs;] . they are gpUjptp [land] , mg;G [water] , NjA [fire] , thA [air] , Mfhak; [ether (or) space] .

Alteration in ratio of gQ;rG+jk in human body leads to vitiation of three humours. Siddha system of medicine is mainly based on the humoral theory, namely Vatha [thjk;], Pitha [gpj;jk;], and Kapha [fgk;]. These three humours are called by different terminologies ie, kyk;> Njh\k;> caph;jhJ> Fw;wk;. Any decrease or increase in ratio of the 3 humours causes disease in human body .

The three humours literally mean wind, bile and phlegm respectively Vatha , Pitha , Kabha are in definite proportions 1: ½ ; ¼.

“toq;fpa thjk; khj;jpiunahd;whfpy;
joq;fpa gpj;je; jd;dp yiu thrp
moq;Fq; fge; jhdlq;fpNa fhNyhby;
gpoq;fpa rPth;f;Fg; gpr nfhd;W kpy;iyNa”

-Fzthfl ehb

The sensory and motor functions of body is based upon 96 principles [jj;Jtq;fs;].

“**Kalanchaga padai**” popularly known as “**Psoriasis**” has been a challenge to the medical world. “**Psoriasis**” and the clinical entity kalanchaga padai are proximate to each other.

Kalanchaga padai is not easily curable. The history of the disease however has been dealt within the siddha system of medicine. Being a social stigma, the disease has now drawn the attention of the scientists all over the world, especially the medical scientists of different systems. People of all groups and races suffer by this disease without any permanent relief .

Moreover the disease can be formed due to changes in the mind also. But “**Siddhars**” also quoted very anciently that many of the diseases were caused by

psychosomatic problems. So that they had advised to control ones mind to get red of stress. This was quoted by “**Agasthiyar**”as follows .

“kdkJ nrk;ikahdhy; ke;jpuQ; nrgpf;f Ntz;lh
kdkJ nrk;ikahdhy; thAit cah;j;j Ntz;lh
kdkJ nrk;ikahdhy; thrpia epWj;j Ntz;lh
kdkJ nrk;ikahdhy; ke;jpuQ; nrk;ik ahNk”

- “mfj;jpah; Qhd ghly;”

The author has taken “**kalanchaga padai**” is their dissertation subject .

It is a disease of physical and mental strain .

Siddhars had explained the line of treatment into 2 major types .One internal medicine and other is external medicine. Both these medicines are 32 in numbers .

“ Nth; ghU jio ghU kpQ;rpdf; fhy;
nky;y nky;y gw;g nre;Jhuk; ghU”

From the above quoting, the author ideally select the purely herbal preparation “**Parangi chakkai choornam**” as her **Internal medicine** and “**Thaser meni ennai**” is the external application.

Besides medicine, diet restriction, special medication [pranayama and Yogasana], were also instructed to the patients. Furthermore, the control of mind is the key to alleviate this skin disease. Information about the research work kalanchaga padai has been described in the forthcoming pages.

AIM AND OBJECTIVES

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The skin is an extraordinary structure and it reflects either healthy or diseased. Kalanchagapadai, which affects not only skin but also produce mental strain. It is a significant disease burden, affecting both health-related and emotional aspects of a patients life. The treatment available for kalanchagapadai at present around the globe does not cure the disease radically. So for such an tractable disease the world craves for a cure.

Siddha science, which gives wonder relief to the skin ailments. It is very open secret in siddha medicine. That's why the author had chosen kalanchaga padai as their dissertation subject in order to make a through research study.

Among all forms of treatment, noi naadal or identification of disease and noi muthal naadal (or) determination of the aetiology of the disease are most important aspects. Once the diagnosis is accurate the treatment may be easily fulfilled,

The main aim and objective of this dissertation include ,

1. To collect authentic measures and review the ideas mentioned in ancient siddha literatures regarding the disease “**Kalanchaga padai** “
2. To create an awareness about the siddha science and highlight the efficacy of siddha drugs among the public.
3. To expose the siddhar diagnostic principles .
4. To know the extent of correlation of aetiology, classification, symptomatology, diagnostic methods and the line of treatment of Dermatology .
5. To make a clinical trial with necessary investigation and records all those things with the follow-up study of the patients of kalanchaga padai.

6. To have an idea of an incidence of kalanchaga pasdai with reference to age, sex, occupation, socio – economic status habits, family history are related to any psycho – somatic problems and paruva kaalams (Seasons).
7. To have a complete study of the disease kalanchaga padai under the headings of
 - a. Poripulungal: C.Enn Vagai thervugal
 - b. Udal kattugal d. Mukkutram etc.
8. To make a detailed clinical evaluation of the disease by careful examination on Aetiology, Symptomatology, Complication, Treatment and Prognosis.
9. To have a clinical trial of kalanchaga padai with the trial drugs **“Parangi chakkai chooranam”** as internal medicine and **“Thaasermeni Ennai”** as the external medicine .
10. To use modern diagnostic parameters to confirm and follow the progress of the patients.
11. To evaluate the bio-chemical analysis, and pharmacological study of the trial drugs.
12. To highlight the factors like land where they live, climate changes, diet, and mental stress of human beings.
13. To make an awareness among the patients inorder to avoid further recurrence of the disease.

REVIEW OF LITERATURES

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A. SIDDHA ASPECTS

“fhshQ;rfg; gil”

NtW ngah;fs;:

nrk;;nghl;L Njhyow;rp> nrjph; cjph;Tg; gil> rhk;gy; gil>

-mUq;fiyr;nrhy; mfu Kjyp

“ntz;gUr;nrjpy;> nrjpy; cjph; Neha;>

- rpj;j kUj;Jtk; rpwg;G

,ay;: (Definition)

Kalanchaga padai is a non-infectious, chronic, recurrent, inflammatory disorder of the skin with reddish, slightly elevated, patches or bumps covered with silvery-white scales , sopts may coalesce into large patches around a normal area.

Neha; tUk; top (Aetiology)

In siddha system of special medicine chronic skin disorders are brought under the clinical entity “kuttam” [Fl;l;]

Among the available siddha literature only “Yugi Muni Vaidhya Chindthamani” and “Thirumoolar vaithiyam “ are the sources of information on aetiology and clinical features of the skin disorders. They classified the “kuttam” into 18 types. There are no specific mention about the specific factors causing kalanchaga padai.

“tpahjpAs; %thW tpsq;fpa Fl;lq;Nfs;

fahjp fpue;jp Roy; Nkfj;jh yhWk;

gahjp kz;Zsg; gy tz;bdh nyl;Lk;

epahjp GOehyha; epd;wjpf; Fl;lNk”

- jpU%yh;

“fpue;jp Rod; Nkfj;jhyhWk;”

1.Six types are caused by venereal origin

“ gahjp kz;Zyg; gy tz;bdh nyl;Lk;”

2.Eight types are caused by insect bites

“epahjp GOehyha; epd;wjpf; Fl;INk”

3. four types are caused by worms infestation.

“gapy; nkhopaPh; jpNufj;jpy; fpUkpjhNd

gue;J jphpFl;lk; Nghy; Gs;sp fhZk;

kayJTk; fpUkpa;e;jh ele;J Gf;fpy;

NkdpaJ rurnud ntbj;J Gz;zhFk;”

-FUehb

“fpUkpahy; te;j Njhlk; ngUfTz;L

Nflfp yjd; gphptjid fpukkhf

ngUkp tUk; thantyhk; fpUkpahNy

GOf;fb Nghy; fhZkJ fpUkpahNy

nrUkp tUk; gTj;jpuq;fs; fpUkpahNy

Njfkjpy; nrhhpf;Fl;lk; fpUkpahNy

JUkp tUQ; RNuhzpjQ; fpUkpahNy

#l;rKld; fphpirg; ghy; njhopy; nra;tPNu”

- FUehb

The text book “Sirappu-Maruthuvam” described the following aetiological factors for “kalanchaga padai”

- VNjhnthU njhpahj fhuzj;jhy; - Unknown aetiology
- guk;giu Nehahf %thpy; xUtUf;Fk; - Genetic Cause

Nehia kpifg;gLj;Jk; fhuzpfs; :- [Triggering factors]

yRdjhgppjk;	-Tonsilitis
Gg;Grg; gpzpf;	-Respiratory disorders
xt;thik	- Allergic disorders
kdcisr;ry;> ftiy	-Stress and strain
mjp;h;r;rp	- Anxiety, Depression
fhykhWghLfs;	- Seasonal variations.
rpY kUe;Jfs;	- Certain drugs

(eg.)

jhk;gpur; nre;Jhuk; - Red Oxide of Copper
FNshNuh Ftpd; - Chloroquin
,sk;gps;is thj jLg;G kUe;J – Polio Vaccine

According to siddha texts the causes of skin ailments[Fl;lk;] are mentioned by the following quotes

“Fl;lKld; jpNufnky;yhk; gwF;Fk;NghJ
FopFopaha; fpUkpadhw; nfhs;Sk; Gs;sp”
-FUehb

“Fl;lkJ tpl fug;ghd; tpl ePh; #iy
RNuhzpjj;jhy; jhJ nfl;L jbg;Gz;lhfK;
kl;lkwNk fpUkp nrd;w kUTk; NghJ
tifaha; fpUkpadl tpl ePh; nrd;W
Fl;lKld; Njfnky;yhk; gwF;Fk; NghJ
FopFopaha; fpUkpadPh;f; nfhs;Sk; Gs;sp
jl;lwNt fpUkpadl ePuhy; te;j
rfy Fl;lk; tpl fug;ghd; rhw;wyhNk”
- jpU%yh;

“Agasthiyar mentioned the following causes for kuttam.

“tpahjpfs; %thWf;Fk; tpsq;fpa Fl;lq;Nfsha;
Rahjpah Nkfj;jhYk; #o;fpd;w fpue;jpahYk;
gahjpah khwjhFk; gy tz;bdhNy nal;lhk;
aahjpahk; GOthdhYk; Iakhk; gjpndl;lhNk”

-mfj;jpah; fhtpak;

“Agasthiyar mentioned that kanmam[fd;kk;]the main cause for kuttam.

In Agasthiyar “kanma Kandan”

“Nrh;e;j Fl;lnkhL FiwNeha;ad; te;j
NrjpNfs; kyuhj tUk;g nfha;jy;
jhhpe;j rPh; nre;J tijfs; nra;jy;
jha; je;ij kdJ nehe;J Nuhfe;jhNd”

-ghly; -76

“jhndd;w nja;tUj; jidaopj;jy;
rhh;thd nghpNahh;fs; jikg; gopj;jy;
fhndd;w ee;jtdk; G+Q;nrbfs; ntl;ly;
fUkkLh rhPuj;jpw; fhR NghNy
A+ndd;w Tlk;ngy;yhk; nkhl;L nkhl;l
Ald; ntSj;J FiwNahAjpUQ; rpe;Jk;
thndd;w fUkq;fs; jPh;g;gjw;f
tiunahd;W nrhy;Ntd; nfs; ee;jtd;ikNa”

- ghly;-77

“In Agasthiyar paripoornam-400”

kanmavaralaru [psycho social cause]

“gotpidahy; tp\g;G+r;rp fbj;j Njh\k;
ghjfh;f;F xU ehSk; jPh;tjpy;iy
cs;tpidahYhlhbf; nfhs;s te;j
cz;ikaJ mwpahky; %h;f;fQ; nra;thh;
fstpidAe; jPh;tjpy;iy fbdnkj;j
fUizAs;s G+uzj;jpd; fz;fhl;rp
mltpid eP fhZKd;Nd mfyr; nrhy;yp
milahsk; tpuy; FWF kpq;dq;NfNs”

-mfj;jpah; ghpG+uzk;-400 nra;As; 214

“tpuy; FWFq;fhy jpkpUk; tp\k; NghNyWk;
nka;aOe;Jk; jiy RoYk; ntSf;F Nkdp
gukhd Njknky;yhe; jbj;J tPq;Fk;
gjnky;yhk; itj;J kpf;F Gz;Z fhZk;
rurKld; nrhhp fug;ghd; gzk; Nghy; NjhZk;
rhe;ijahNk tpe;ijnfLj; jbj;J tPq;Fk;
ghUyfp ype;Neha;f;F kUjPahNj
ey;Nyhiug; gopj;j Fl;lq;fs; d;dkhNk”

-mfj;jpah; ghpG+uzk; -400 nra;As; 215

gotpidahy; tUk; Neha;fs; jPh;tjpy;iy vdTk;> fd;kk; jPu Ntz;Lk; vd;Wk;
mfj;jpah; ghpG+uzj;jpy; \$wg;gl;Ls;sJ.

In Yugi-800, Yugi described the following causes of kuttam

- Close contact with the diseased persons
- Excessive intake of fish, snail, crab etc.
- Usage of things which are used by the diseased persons.

- Doing yoga practice immediately after intake of diet
- Excessive chillness, excessive hot, excessive sleeping, mental stress
- Routine diet, sometimes combined along with unwanted things like sand, hair etc.

In Yugimuni 800, Yugi also mentioned the following causes for kuttam.

“Mr;nrd;w gjpndl;L Fl;le;jhDk;
mtuth;fs; nra;fpd;w mjh;kj;jhyhk;
njr;nrd;w rpthyaj;jpYr; rpl;lq;fs;
nra;jth;fs; rpt epe;ij gz;zpdhh;fs;
%r;nrd;w nghpNahiu Jbj;Njhh;fs;
%h;f;fkha; milf;fyj;ij vLf;fpd;whh;fs;
Mr;nrd;w jpizastpd; Fiwe;j \$yp
nfhLf;fpd;Nwhh; Fl;l;j;jpw; \$LthNdh”

- A+fpKdp-800 ghly; 496

Yugi described only psycho-social factors as the main causes. They are stress inducing factors.

According to “Yugi Vaidhya chinthamani” Yugi classify kuttam into 18 types.

“Kj;jhd Fl;le;jhNd gjpndl;Lf;Fk;
Kdpahd A+fp ehd; nrhy;yf; Nfsha;
gj;jhFk; Gz;lhpff; Fl;l;j;NjhL
nghUfpd;w tpw;Nghlf Fl;lkhFk;
gj;jhFk; ghkf;Fl;lk;> frrh;kFl;lk;
ghpthd fh;zFl;lk;> rpFuFl;lk;
fpj;jhFk; fpUl;bzf;Fl;lk;> mTJk;guf;Fl;lk;
nfbahd kz;lyFl;lKkhnkd;Nd

Fl;lkhk; gg;ghpr Fl;lnkhL
Fbykhk; tprh;r;rpff; Fl;lkhk;
tl;lkhk; itahjp Fl;lnkhL
kUtyh fPBg Fl;l rh;kjy
jpl;lkhe; Njj;jpU Fl;lnkhL
rpj;Jkh> Fl;lk;> rjhU Fl;lk;
Jl;lkhU Fl;le;> jd;ndhnlhf;f
Rak;ghd gjpndl;L Fl;lchr;Nr”

- 1.Pundareegam-padar thamarai
2. Virpodagam-Koppulam
3. Bamam- Sirangu
- 4.Gaja sarmam- yaanai thol
5. Karnam-Kaadhu
6. Sikuram-Tholperunoi
7. Krishnam-Karuperunoi
8. Avudhumbaram-Athikkai
9. Mandalam-Valayam
10. Abarisam- Vali
- 11.Visharchigam-Sori
- 12.Vibhadhigam-Senkuttam
- 13.Sarmathalam-Tholvedi
- 14.Kideopam-Pantrithol
- 15.Thathuru-Thadippu
16. Sithma-Naa
- 17.Sadharu-purai
18. Suvedham-venkuttam

In Yugi chinthamani among the eighteen types of skin (kuttam) diseases, clinical features of three types resemble to that of kalanchaga padai. But no description is available in the siddha literature under the heading kalanchaga padai.

The three types of kuttam are

1. Thethuru kuttam
2. Gajasarma kuttam
3. Virpodaga kuttam

1. Thethuru Kuttam

“rh;ke;jhd; rptg;ghf tl;lzpj;Jr;
ryit Nghy; ntSf;FNk jpdTz;lhFk;
th;ke;jhd; NuhfkJ kpfTz;lhFk;
kapnuy;yhQ; RUz;LNk cz;ilahFk;
fh;ke;jhd; gpj;j Nrl;Lk kpFf;Fk;
fhae;jhd; fjpj;JNk jpkpUz;lhFk;
jh;ke;jhd; rlnky;yh %jyhFk;
jhf;fhd Njj;jpUf; F\;le;jhNd”

A+fp Kdp itj;jpa rpe;jhkzp 800> ghly; 511

Under “Thethru kuttam”, annular erythematous lesions with the white appearance, itching, oedema of the body and rolling of hair like balls are the characteristic clinical features in this entity.

2. Gajasarma Kuttam

Clinical features:

“jhdhfr; rle;jhD kpff; fWg;ghk;
rlnkq;Fe; NjhYhpAQ; rptg;GkhFk;

Ntdhd ntw ntwdj; jhdPOf;Fk;
ntbf;FNk nrhhpr;ryha;j; jpdTz;lhFk;
fhdhfr; rh;kFl;lk; kpjpYz;lhFk;
fbdkha;f fhy; tpuy;fs; fdg;Gz;lhFk;
\$dhfj; Njfnkq;Fk; typnaLf;Fk;
Fwpahd frfh;k Fl;le;jhNd”

Blackish discolourations of the body, peeling of skin, itching, erythematous lesions , oedema of the toes, body pain are the clinical features of Gajasarma kuttam .

3.Vithpodaga Kuttam

Clinical features:

“GJikaha;r; rhPunkq;Fe; jpdTz;lhFk;
nghUntbaha;j; jpf;nfdj;jPf; nfhOe;J Nghy
nkJikaha; tpl;nlhpAk; ey;y ghk;gpd;
tp\g;glk; NghNy jbj;J ntSg;G khFk;
fJikaha; kpfr; nrhhpAQ; rptg;GkhFk;
Jhf;fnkhL rQ;ryKk; kpFTz;lhFk;
fJikaha; Njhnyy;yhe; jbg;Gz;lhFk;
fdj;jtpw; Nghlfkhd Fl;le;jhNd”

A+fp Kdp ngUEhy; 800> ghly;-498

A skin erythematous lessions with swelling , white appearance and itching are described.

Usually these entities are associated with anxiety and despair.

In the text book “**Siddha Maruthuvam Sirappu**” author by **Dr.R.Thiagarajan. LIM;** describes, the general clinical features of “kalanchaga padai” as follows,

1. The skin lesions are patches and macules which are red in color and raised margin and the lesions are covered by silvery, white and rough thick scales, pin point bleeding after rubbing of scales.
2. There are variations in the size and shape of patches according to the site.
3. In children these lesion may be like water drops and these may occur in scalp and face. Sometimes as per the severity it seems all over the body.

In chronic Cases:

1. The skin lesion occur over the front of and back of elbows,
2. In some, these patches appear over the palm and soles.
3. In some, the patches occur all over the body with excessive scaling.
4. The patches are coin shaped over them. The shape may be either round or oval.
5. In obese women the lesion may occur over inguinal axillary, naval regions and folding areas with wet.
6. One fourth of patients have lesion over nails pitting and thinning in nature
7. 7% of patients develop affection of joints as psoriatic arthropathy.

Arthropathy (*fhshQ;rf thjk*;)

Kalanchaga padai is often associated with painful joints [Arthritis] is known as **“Kalanchaga vatha”**. In few cases arthritides incapacitates the patients to resort to hospitalization. Any joint may be affected. The most often affected joints are interphalangeal joints.

The terminal interphalangeal joints are usually involved as opposed to the proximal interphalangeal joints in **“Vali azhal keel vayu”** which is identical with rheumatoid Arthritis. In these cases the affected fingers shows nail changes. This combination is termed **“Psoriatic arthropathica”**

The joints of fingers, ankles, knee and sacroiliac are selectively affected. Those joints are swollen and painful with psoriatic lesions. Radiological changes shows gross changes in the affected area. These are characteristics and consists of osteoporosis

followed by decreased density, diminished joint space, erosion of joint surfaces followed by eventual destruction of the end bones.

Yugi muni describes the clinical features of kalanchaga vatham as follows.

“khjkhq; fhy; ifapy; Fuq;fpuz;Lk;
tUj;J re;J KWf;fpNa File;J nehe;J
ehjkh eil jhDe;jhd; nflhky;
eype;JNk Klkhfpf; fuL fl;br;
NrjkhQ; rle;jhD kpf ntSj;Jj;
jpdNthL rpuq;Fkha;r; Nrl;gkhfpf;
fhjkh aUrpnaL kaf;fkfFk;
fUjpNa fhshQ;rfkhk; thjkhNk”

Fuq;F	- njhil
re;J	- ,Lg;G
Filr;ry;	- tUj;jk;> czT
eypj;jy;	- Jd;gg;gLj;jy;
Klk;	- neh;zb
fuL	- fZ
jpdT	- nrhwp
rpuq;F	- xU FU Neha;

“khjkhq; fhy; ifapy; Fuq;fpuz;Lk;
tUj;J re;J KWf;fpNa File;J nehe;J”

The joints of fingers, feet, ankles, knee and sacroiliac are selectively affected and these joints are painful.

“ehjkh eil jhDe;jhd; nflhky;
eype;JNk Klkhfpf; fuL fl;br;”

The deforming erosive arthritis targets fingers and toes. Marked cartilage and bone attrition results in loss of joint and markedly instability. Bone erosion and destruction take place leading to collapse of affected digits.

“Nrkhe; rle;jhD kpf ntSj;Jj;
jpdNthL rpuq;Fkha;r; Nrl;gkhfpf;
fhjkh aUrp nahL kaf;fkhFk;
fUjpNa fhshQ;rfkhk; thjkhNk”

The whole body becomes pale (Anaemic). A well-defined erythematous papules which are sharply demarcated appear on the skin. There is also loss of taste and giddiness.

nghUSiu:

fhshQ;rfk; vd;gJ vz;gJ tif thj Neha;fSs; xd;W. ,jdhy; fhy; iffspd; %l;Lfspy; KWf;fp typ
nfhLf;Fk;. elf;fKbahJ. fuL fl;b Kl;f;Fk;. cly; kpfTk; ntSj;Jf; fhzg;gLk;. rpuq;Ffs; cz;lhFk;. mUrpAld; kaf;fKz;lhFk; ,ay;Gila Neha;.

SIDDHA PATHOLOGY

NOI (PINI) :

Definition:

Whenever alterations occur in three vital humours disease (pini) or noi occurs.

“clYld; gpize;j caph; mDgtpf;Fk;
,d;g czh;r;rp;F khwhd czh;r;rpia gzp”

An alteration in three vital humours may occur due to

1. Dietary habits
2. Seasonal variations
3. Environment conditions
4. Suppression of 14 urges

5. Altered udal thathukkal
6. Immoral Activities.

Thiruvalluvar Says,

“kpfpDk; FiwapDk; Neha;nra;Ak; EhNyhh;
tsp Kjyh vz;zpa %d;W”

Therefore the dearranged vatha, pitha and kapha denotes disease. The diseases are reflected through the pulses in the three humours.

gpzpfspd; Kjw; fhuzk;

“thj kyhJ Nkdp nflhJ – tsh;gpj;je;
jPjyhJ rj;jpalhJ – Nrj;Jkj;jpd;
NfhjkyhJ tpf;fnylhJ – Fly; jd;dpy;
rPjkyhJ RuKk; tuhJ – jPukhNk”

“ke;jkyhJ thAtuhJ – mdy; gpj;jj;
njhe;jkyhJ %yk; tuhJ – njhlh;thj
ge;j kyhJ Fd;kk; tuhJ – gfh; gpj;j
tpe;ij ayhJ Nkfk; tuhJ – jPukhNk”

“mrPh;zkd;wpr; Ruk; tuhJ – jphpNjhlf;
fhuz kpd;wpr; re;jp tuhJ – fgkhd
ePuJTkp;wpr; Nrigh tuhJ neLthjr;
rhh;tJTk;wpr; #iy tuhJ jtwNj”

,J jtpu gpzpaZfh tpjpapy; \$wg;gl;Ls;s xOf;fq;fis filg;gpbff;f jtWk; NghJ Neha;
cz;lhfpwJ.

FOOD VARIATIONS:

“ Gsp Jth; tpQ;Rq;fwp ahh; G+hpF;Fk; thjk;
Ysp Ath; ifg; Ngwpy; gpj;Jr; rPWk; - fpSP nkhopNa
fhH;g; gpdpg;G tpQ;rpw; fgk; tpQ;RQ; rl;bujr;
Nrug; Gzh; NehaZfhNj”

Sour and astringent increase vatham

Salt and bitter increase pitham

Purgent and sweet increase kabam

ENVIRONMENTAL CHANGES:

Environmental means place of living (nilangal). It is divided in 5 major types. They are kurinji, mullai, marutham, neithal and palai. The place of living is important because certain diseases are predominant only in certain places.

Kurinji-Mountain and its surroundings

Mullai-Forest and its Surroundings.

Marutham-Fertile land and its Surroundings.

Neithal-Sea shore and its Surroundings

Palai-Desert and its Surroundings

NILAM	HUMOURS	DISEASE
Kurinji nilam	Kabam	Abdominal tumour, fever
Mullai nilam	Pitham	Vatha diseases
Marutha nilam	All three humours are in the equilibrium	Nil
Neithal Nilam	Vatham	Increasing body weight,

		Havpatomegaly, splenomegaly
Palai Nilam	All three humours are affected	Genesis of vatha, pitha and kabam diseases.

PARUVA KAALAM (SEASON)

One year is classified in to six seasons. Each season constituting 2 months.

SI No	SEASONS	MONTHS	HUMOURS
1	Karkalam	Avani-puratasi (Aug 16 – Oct-15)	Vatham↑↑ Pitham↑
2	Koothir Kalam	Iypasi-Karthigai (Oct 16-Dec 15)	Vatham → pitham↑↑
3.	Munpani kalam	Markazhi-Thai (Dec 16-Feb15)	pitham→
4.	Pinpani Kalam	Masi-Panguni (Feb15-Apr 15)	Kabam↑
5.	Ilavenir Kalam	Chithirai-Vaikasi (Apr 16-June 15)	Kabam↑↑
6.	Mudhuvenir Kalam	Aani-Aadi (June16-Aug 15)	Vatham↑ Kabam→

↑↑ Vettrunilai valarchi

↑ Thannilai valarchi

→ Thanilai adangal

FOURTEEN VEGAMS:

Self-suppression of fourteen vegams causes diseases.

1. Vatham: Downward Force:

Heart diseases, vath gunmam, kudal vatham, vali vatham, constipation, loss of appetite.

2. Sneezing (Thummal)

Head ache, special sense organ defect, facial pain.

3. Siruneer (Urine)

Urine retention, Urethral mulcer, joint pain, urine micturition pain in the penile part, flatus abdomen.

4. Malam (Faecus)

Diarrhea caused by increased abanan, knee pain, headache, weakness.

5. Kottavi (Yawning)

Lethargic face, exhaustion, indigestion, Leucorrhoea, neernoi, abdominal disease, Loss of consciousness.

6. Hunger and Thirst:

All organs are affected amacication, soolai noi, apathicface, joint pain.

7. .Kasam

Increased cough, bad breathe, heart diseases.

8. Elaippu(Exhaustiveness)

Peptic Ulcer, neer megham, rigor, syncope.

9. Thookam (sleep)

Heaviness of head, eye diseases, deafness, confused speech

10. Vanthi (vomiting)

Utricularial rashes, itching sensation, eye diseases, asthma, fever, cough.

11. Kanneer(Tears)

Hear diseases, sinus diseases, eye diseases, necrotic ulcers in the scalp, peptic ulcer.

12. Sukkilam

Fever, urine retention, joint diseases, chest pain, Leucorrhoea.

13. Suvasam (breathing)

Cough, abdominal discomfort, fever, venereal diseases, anorexia.

Udal Kattugal

Our body consists of seven udal kattugal. It gives strength and structure to our body.

Sl no	Udal kattugal	Functions
-------	---------------	-----------

1.	Saaram	It gives strength to the body and mind
2.	Senneer	Saram after absorption is converted into senneer. It is responsible for knowledge strength, boldness and healthy complexion.
3	Oon	Gives structure and shape to the body and is responsible for the movement of the body.
4.	Kozhuppu	Lubricates the organs and proceed on its own works
5	Enbu	Protects the vital organs and used for movements and nominates body structure.
6	Moolai	Present inside the bones and it gives strength and maintain the normal condition of the bone
7	Sukkilam (or) suronitham	Responsible the reproductive function of species.

UDAL KATTUKKAL

SL NO	UDAL KATTUKKAL	DECREASED FEATURES	INCREASED FEATURES
1.	SAARAM	<ol style="list-style-type: none"> 1. loss of weight 2. lassitude 3. Dryness of the skin 4. Diminished activity of the sense organ 	It leads to disease like loss of appetite, profuse salivation, depression etc.
2.	SENNEER	<ol style="list-style-type: none"> 1.Tiredness 2.Lassitude 3. Anaemia 4. Dryness 	<ol style="list-style-type: none"> 1.increased blood pressure 2.Reddish eye and skin 3. Jaundice 4.haematuria 5. Boils and tumours in different parts of the body
3	OON	<ol style="list-style-type: none"> 1.Muscle wasting 2.Lethargic sense organs 	1.Extra growth around neck, face, abdomen, thigh, genitalia etc.,

In the case of kalanchaga padai out of seven udal kattukkal saaram, senneer, oon and enbu are commonly affected.

Saaram : Dryness, roughness, tiredness

Senneer : Dryness, paleness of the skin

Oon : Weakness of sense organ.

Enbu : pain in the joints in chronic cases

Mukkutram :

Human body is influenced by three doshas such as vatham, pitham and kabam. They are responsible for normal physiological condition of the body.

Vatham:

Vatham is a kinetic energy, which influences all movements. Vatham is located in the abanan, idakalai, faces, spermatic cord, iliac bone, skin, nerves, joints, hair follicles, muscles, bone, ear and thigh.

Vatham classified into 10 types. They are,

Sl no	Name	Locations	Physiologic functions
1.	Piranan	Heart and lower and Upper respiratory Tracts	Controls knowledge, mind and five objects of sense for breathing
2.	Abanan	Lower abdomen and extremities	Responsible for urination, expels faces and foetus, discharge sperms and menstruation
3,	Viyanan	Mainly at heart	Responsible for movement of all parts of the body and used to feel the sensation
4.	Uthanan	Chest	Responsible for vomiting cough, hiccough, sneezing
5	Samanan	Stomach	Aids for proper digestion. It controls the activity of other vayus.
6	Naagan	Eyes	Responsible for opening and closing of the eyes
7	Koorman	Heart and Eyes	Responsible for vision and yawning and controls lacrimation
8	Kirukaran	Throat	Responsible for salivation nasal secretion and appetite
9	Thevathathan	Eruvai & Karuvai	For laziness, sleeping and anger
10	Thananjeyan	Nose	Responsible for bloating of the body after death. It escapes on the third day after death through the cranium when it bursts.

In the case of kalanchaga padai

1. abanan – Habitual Constipation

2. Viyanan – Erythematous in the affected lesions of skin
3. Samanan –due to other vayus it is affected
4. kirukaran- polydipsia, polyphagia, loss of appetite.
5. Devathathan – insomnia like condition

The above vayus are affected commonly.

Pitham :

Pitham is responsible for all the transformation. Pitham is located in urinary bladder, heart, head, umbilicus, abdomen, blood, sweat, skin and eye.

Pitham is classified into 5 types, they are,

1. Anala pitham –responsible for digestion of food
2. Ranjaga pitham- responsible for colour of blood
3. Sathagam –located in heart and is responsible for normal activities of the body.
4. Alosagam- Responsible for normal vision
5. Prasagam- Responsible for the complexion of skin

In case of kalanchaga padai

1. Anala pitham – Indigestion of food.
2. Ranjagam – Paleness of the conjunctiva and tongue
3. Sathagam- Difficulty to do the routine works properly and sluggishness
4. Pirasagam – Dryness and roughness of skin.

Kapham :

Stabhizes maintain and lubricates all movements. Kapha is located in samanan, semen, head, marrow, blood, nose, chest, nerves, bones, brain, large intestine, eye and stomach. Kapha is classified into 5 types. They are,

1. Avalambagam : Heart is the centre of avalambagam. It controls all the other kaphas.

2. Kilethangam: Stomach is the center of kilethagam. It gives moisture and softness to the ingested.
3. pothagam: Tongue is the centre of pothagam, responsible for the sense of taste.
4. Tharpagam: Head is the centre of tharpagam gives cooling effect to the eyes.
5. Santhigam : It lies in the joints and responsible for the action of joints.

In case of kalanchaga padai,

1. Kilethagam-loss of appetite was mainly affected.
2. Tharpagam- Burning sensation of eyes was affected in few cases.
3. Santhigam- pain in joint affected in few cases.

MUKKUTTRAM

SL NO	HUMOURS	INCREASED	DECREASED
1.	VATHAM	Body pain, pricking pain, nerve weakness, joint pain, mental distress, dislocation of joints of upper and lower limbs, Anrexia, constipation, difficulty in flexion and extension of limbs, all taste like astringents, darkness of motion and urine	Body pain, Aphasia, Confusion, Dyspnoea, loss of body activity

2	PITHAM	Yellowish discoloration of eye, skin, urine, faeces polyphagia, hunger, burning sensation all over the body, sleeplessness, cough, dyspnoea, excessive sleep	Decreased appetite, symptoms associated with defective growth, kabam, cold, pallor.
3	KAPHAM	Diminished activity heaviness, cough, dyspnoea, excessive sleep	Giddiness, prominence of bone edges, dry cough, lightness excessive, sweating, palpitation.

RELATION BETWEEN SUVAI, PANCHABOOTHAM AND MUKKUTRAM.

SL NO	Suvai	Panchabootham	Mukkutram
1.	Enippu(sweet)	Piruthivi+Appu	Kapha↑ Vatha↓(-) Pitha↓(-)
2	Pulikppu (sour)	Piruthivi+Theyu	Kapha↑ Pitha↑ Vatha↓(-)
3	Uppu (Salty)	Appu+Theyu	Kapha↑ Pitha↑ Vatha↓(-)
4	Kaippu (Bitter)	Vayu+Space	Vatha↑ Kapha↓(-) Pitha↓(-)
5	Karppu(Pungent)	Vayu+Theyu	Vatha↑ Pitha↑ Kapha↓(-)
6	Thuvarppu(Astringent)	Piruthivi+Vayu	Vatha↑ Kapha↓(-) Pitha↓(-)

↑ - Valarchi

↓ - Samanappaduthal

UDAL VANMAI-BODY IMMUNITY:

The udal vanmai is classified into 3 types, they are,

1. Iyarkai Vanmai
2. Seyarkai Vanmai
3. Kaala Vanmai

1. Iyarkai Vanmai:

Natural immunity of the body itself by birth

2. Seyarkai Vanmai:

Improving the health by intake of nutritious food materials, activities and medicines.

3. Kaala Vanmai:

Development of immunity according to age and the environment.
When Udal vanmai is affected there may be a possibility of kalanchaga padai.

GNANENTHIRIYAM:

Gnanenthiriyam are Mei, Vai, Kan, Mooku, Sevi.

- 1 Mei – feels all types of sensation
2. Vai- for recognize taste
3. Kann-Meant for vision
4. Mooku- for recognize smell
5. Sevi-for hearing

Incase of “kalanchaga padai”

1. **Mei** : Roughness of the skin, white silvery scales are affected generally, other are not affected.

KANMENTHIRIYAM:

Kanmenthriyam are kai, kaal, vaai, eruvaai and karuvai.

1. Kai – majority of normal works done by hands
2. Kaal- for walking
3. Vaai-For speaking
4. Eruvaai- for defaecation
5. Karuvaai-for reproduction

In case of “**kalanchaga padai**”

1. Kai, Kaal- Difficult to use the limbs in this stage of kalanchaga vatham.

PINIYARI MURAIMAI

(Diagnostic methods adopted in siddha system of medicine)

Piniyari muraimai is the methods of determination of a disease.It is based on the following principles.

1.Poriyalarithal

2.Pulanalarithal

3.Vinaathal

“Pori” are the five organs of perception namely.

Nose

Tongue

Eyes

Ears

Skin

“Pulan”are the five object of senses namely.

Smell

Taste

Vision

Auditory

Sensation

Alavaigal are uses in clinical diagnose of a disease

“ msit fhz;ly fUjy; ciu mghtk; nghUs; xg;ghnwd;gh;
msit NkYk; xopGz;ik iajpgfj; Njhbay; ngd ehd;
fsit fhz;gh; mitapw;wpd; NkYk; miwth; mitnay;yhk;
msit fhz;ly; fUjy;> ciu vd;Wk; %d;wpylq;fpLNk.

- rptpjj;pahh; msit vz;.6

Alavaigal is divided into ten types they are

- | | |
|------------------------------------|------------------------------|
| 1.Observation – fhz;ly; | 6.Comparison - cgthdk; |
| 2.Inference - fUjy;
elimination | 7.Inference by - ghhpNr\k; |
| 3.Authority, - ciu
Literature | 8.Probability - rk;gtk; |
| 4. Perception - mghtk; | 9.Tradition - IjPfk; |
| 5.Presumption – mUj;jg;gj;jp | 10.Natural Inference - ,ay;G |

Poriylarithal and pulanalarithal goes hand in hand with the concept of examining the patient's **pori and pulan** with that of **physician's pori and pulan**.

Vinaathal is a method of enquiring about the details of that patient's problem from his own words or from his parents or neighbours who are taking care of the patient, when the patient is not able to speak (or) patient may be a child.

This principle can be compared to that of interrogation and inspection in modern Aspect. Besides this, "**thottu parthal**" (palpation) and "**thatti parthal**" (percussion) are also used to examine the patients.

Envagai Thervugal:

The siddha system is very precise methods for understanding the disease processes before any overt signs of the disease have manifested. By detecting early symptoms of imbalance and disease reaction in the body one can determine the nature of future bodily reactions. Observation of envagai thervugal indicates what pathological changes are occurring in the body. Which organs are impaired and which are the doshas accumulated.

Thus by checking the body's indicators-regularly, pathological symptoms can be detected early and preventive measures are taken early.

vz; tiff; Njh;Tfs; gw;wp:

1. “nka;f;Fwp epwe;njhdp tpopehtpU kyk; iff;Fwp”

nka;Fwp	: Signs in the body
epwk;	: Colour
njhdp	: Sound and speech
tpop	: Eye
eh	: Tongue
,Ukyk;	: Urine & Stool
iff;Fwp	: Signs in hand-Pulse

1. ehb ghprk; eh epwk; nkhop tpop
kyk; %j;jpuk; kpit kUj;Jt uhAjk;”

1. ehb
2.];ghprk;
3. eh
4. epwk;
5. nkhop
6. tpop
7. kyk;
8. %j;jpuk;

3. juzpAs;s tpahjpfis al;lhq; fj;jhy;
jhdwpa Ntz;LkJ VNj njd;wy;

jpuzpa Njhh; ehbfzpf; rj;jj;NjhL
 Njff;jpdJ ghprk; tz;zk; ehf;F
 ,uzkyk; %j;jpukhk; ,itfnsl;lhk;
 ,jk;glNt jhd; ghh;j;Jf; Fwpg;gha;f;fz;L
 gudUshd; nghpNahh;fs; ghjk; Nghw;wpg;
 gz;Gjt wjgz; jQ; nra;tPNu”

mfj;jpah; Fzthfl ehb

“njhFf;fYw;w ml;l tpjg; ghPl;ir jd;id
 Jyf;fKWk; gz;b;Nu njspthfg;
 gFf;fhpa ehbia eP gpbj;J ghU
 gfh;fpd;w thh;j;ijiag; ghh; ehitg;ghU
 tFf;fhpa Njfnkd njhl;Lg;ghU
 tskhd rhPuj;jpd; epwj;ijg;ghU
 rfpf;fhpa kyj;ijg; ghU ryj;ijg;ghU
 rhh;e;j tpopjidg; ghh;j;J njspthff; fhNz”

- mfj;jpah; itj;jpa ty;yhjp-600

Naadi	-	Pulse
Sparism	-	Sensation
Naa	-	Tongue
Niram	-	Colour
Mozhi	-	Sound
Vizhi	-	Eye
Malam	-	Faeces
Moothiram	-	Urine

The speciality of eight tools of diagnosis is mentioned in the following

Verses also.

“ePba tpopapdhYk; epd;w ehf;Fwpg;gpdhYk;
thba NkdpapdhYk; kynkhu ePhpdhYk;
#ba tpahjpjd;idr; Rfk; ngw mwpe;J nrhy;Ny”

Kalanchaga Padai in relation with Ennvagai thervugal

1.Naadi (Pulse)

clypy; caph; jhpj;jpUg;gjw;Ff; fhuzkhd rf;jp vJNth mJNt jhJ my;yJ ehb vd;gLk;.

“ehb vd;why; ehbay;y> euk;gpy; jhNd
eykhfj; Jbf;fpd;w JbjhDky;y
ehb vd;why; thj gpj;j rpNyw;gdKky;y
ehb vOgj;jPuhapue; jhDky;y
ehb vd;why; mz;l Nguz;lnky;yhk;
ehb vOtifj; Njhw;wj;Js;sha; epd;w
ehbaJah uha;e;J ghh;j;jhuhdhy;
ehbAWk; nghUs; njhpe;J ehL thNu”

Naadi is reflexing the vitiating elements of the body which are Vatha, Pitha and Kapha and can be felt one inch proximal to the wrist on the radial side by means of Palpation with the tips of index,middle and ring fingers corresponding vatham,pitham and kapha respectively.

The three humours vatham,pitham and kabam exists in the ration 1:1/2:1/4 normally.Derangement in these rations leads to various disease entities.

The three “Uyir thathukal” are formes by the combination of three nadigal with three Vayu

- a) Edakalai + Abanam = Vatham
b) Pinkalai + Piranan = Pitham
c) Suzhumunai + Samanan = Kapham

In kalanjaha padai the following types of naadi can be see commonly.

They are,

- a) Vatha pitham
b) Vathakabam

II.Sparism

In case of Kalanjaga padai well defined macules,papules,thickening,roughness,pain and white silvery scaling of skin can be noticed at affected area.

III.Naa

In case of Kalanchaga padai no abnormality is seen in Naa.

IV.Niram

In case of Kalanchaga padai white patches with silvery scales can be noticed at affected areas.

V.Mozhi

In case of kalanchaga padai,no abnormalities was ruled out.

VI.Vizhi

In case pf Kalanchaga padai,no abnormality is seen in vizhi.

VII.Malam

In case of Kalanchaga padai constipation was reported in some cases.

VIII.Moothiram

Collectin of urine for the determination of Neerkuri and Neikuri, a special Diagnostic method.

Neerkuri and Neikuri

“mUe;J khwpujKk; mtpNuhjkjha;
m/fy myh;jy; mfhyT+d; jtph;e;jow;
Fw;ws tUe;jp cwq;fp itfiw
Mbf; fyrj; jhtpNa fhJnga;
NjhU K\$h;j;jf; fiyFl; gLePhpd;
epwf;Fwp nea;f;Fwp epUkpj;jy; flNd.”

-rpj;j kUj;Jthq;f RUf;fk;

Prior to the day of urine examination the patient is instructed to take a balanced diet. The patient could have good sleep. After waking up in the morning, the first urine voided is collected in a clear wide mouthed glass container and is subjected to analysis of “neerkuri and neikuri” with in one and a half an hour. Then neerkuri is to be found out by

Neerkuri

“te;j ePh;f;fhp iail kzk; Eiu vQ;rnyd;
iwe;jpa Ysit aiwFJ KiwNa”

-rpj;j kUj;Jthq;f RUf;fk;

Voided urine has the following characters

- 1.Niram - Colouration
- 2.Edai - Specific gravity

- 3.Manam - Smell
- 4.Nurai - Froth nature
- 5.Enjal - Quantity of urine voided

Apart from these,the frequency of urination,abnormal constituents,such as sugar, protein,presence of blood,pus,renal crystals also to be found out.

In Kalanchaga padai patient straw coloured urine is noticed.Polyurea can be noted in some cases.

Neikuri:

The speciality of neikuri is stated in the following verse.

“ epwf;Fwpf; Fiuj;j epUkh zePhpw;
rpwf;f ntz;nza; Nahh; rpWJsp eLtpLj;
njd;Wwj; jpwe;njhyp Nafhjik;jjp
dpd;wjptiy Nghk; newptpopawpaTk;
nrd;wJ GfYQ; nra;jpia AzNu”

-rpj;jkUj;Jt Neha; ehly; Neha; Kjdhly; jpul;L

The collected specimen as said above is to be analysed by following method.The specimen is kept open in a glass dish or china clay container.It is to be examined under

direct sunlight,without any shaking of the vessel.Then add one drop of gingely oil through the side of vitreous without disturbing the urinary specimen and the neikuri was noted in direct sunlight, and conclude the diagnosis as follows:

Character of vatha neer.

“munt d ePz;bd/Nj thjk;”

When the oil drop lengthens like a snake, called as “ thj ePh; “

Character of Pitha neer.

“Mop Nghy; gutpd; m/Nj gpj;jk;”

When the oil drop spreads like a ring, called as “**Pitha neer**”

Character of Kaba neer

“Kj;njhj;J epw;fpd; nkhopt njd; fgNk”

The oil drop in kapha neer resembled like a pearl. In the Kalanchaga padai the neikuri shows like a “Pearl”.

MUKKUTRA PATHOLOGY

Basically skin is composed a **“Mann and Vali boothas”** as its main components. The sensory aspects and peripheral circulation are attributed to “Viyanan vatha”. Precisely speaking all the three kuttras viz, vali, and iyyam are affected at sub-acute level without producing any serious symptoms for the existence of the affected individual.

The primary humor leads to the disorders of functions of certain types of vayus like abanan, udanan, viyanan, samanana, nagan, koorman and devathathan. Involvement of viyanan affects movements in the various parts of the body, circulation, motor and sensory functions etc. Involvement of abanan leads to constipation.

In seven udal thathus, saaram and senneer are affected leading to lethargic and depressed conditions. Senneer when affected leads to nervousness, dryness of skin, diminution of body lusture.

Among the mukkutram vatha and pitha are mainly affected and lead to some extent. In terms of Siddha stress is a manifestation of provoked pitha. So the provocation may affect the prasaga pitha which is responsible for the nature of the skin, its moisture and complex with erythematous skin lesions. Due to involvement of vatha, there is pronounced itching and some cases involvement of joints. Due to involvement of kapha skin and its appendix are affected. There is an excessive formation of scales and nails deformity in chronic stage. Viyanan, santhigam and kapha are potentially affected and cause serious structural and functional changes in the joints, may occur in serve chronic cases.

Paripoorna Naadi

2. ghh;gpj;j Nkfnkd;why; gpj;j kPwk;
ghyfNd fq;if nfhz;L ePuhk;ghNu

.....

tpuzKld; Gz;Giuf;F thjgpj;jk;

Kavi Naadi

“thA kpFe;jjdhy; tsh;e;Nj jPUk; tpuznky;yhk;

.....

Vaithya sathaga naadi

“jhdKs;s Nrj;Jkk;> jhdpsfpy; ntg;g

rak;> <is ,Uky; ke;jhu fhrk;

<dKWQ; rd;dp tpINjhlk; tpf;fy;

,Uj;Nuhfk; fug;ghd; tpuz Njhlk;

Guru naadi

“nghUkp tUk; tha;nty;yh fpUkpahNy

jpNufj;jpy; nrhhp Fl;lk; fpUkpahNy”

“(gpzp ePf;fk; - ghpfhuk;) – Line of Treatment)”

“cw;wtd; jPh;g;ghd; kUe;Jior; nry;thndd;
wg;ghdhh; \$w;Nw kUe;J”

- jpUts;Sth;

In siddha system of medicine, the main aim of the treatment is to cure udalpini (due to changes in Mukuttram) and Manapini (due to changes in Mukkunam). Treatment is not only for perfect healing but also for the prevention and rejuvenation.

cw;whdsTk; gpzpasTq; fhyKk;

fw;whd; fUjpr; nray;”

“Neha; ehb Neha; Kjy; ehb mJ jzpf;Fk;

tha;ehb tha;g;gr; nray;”

- jpUts;Sth;

Thiruvalluvar says in “**Thirukkural**” about physician’s duty to study the disease, study the cause, seek subsiding ways and do what is proper and effective.

So it is essential to know about the disease, the aetiology, the nature of the patient, severity of the illness, the seasons and the time of occurrence must be observed clearly.

Line of treatment is as follows:

Kappu(Prevention)

Neekkam(Treatment)

Niraivu(Restoration)

Kappu: (Prevention)

As per siddha system,even during the time of conception the vinai payan (tpidg;gad;) in transferred into the fertilized embryo,which is aetiology for certain diseases like, “Kalanchaga Padai” The diseases may be cured not only by medicines but also by teaching the following habits.

- 1.Taking laxatives once in 6 months.
- 2.Avoid stress and strain.
- 3.Teaching good moral habits.
- 4.Always have good mental thoughts by doing meditation.
- 5.Yoga

Skin is the reflex of mind and so we should treat not physique but also treat mind and soul.

“kdjpw;F kUj;Jtk; ghh;f;f Kayhky;
clYf;F kl;LNk kUj;Jtk; ghh;f;f KaYtJ
kUj;Jth; nra;Ak; kpfg; ngUk; jtwHfK;
cs;sKk; clYk; ,iz gphpahjit. ,uz;ilAk;
gphpj;Jj; jdpj;jdpNa rpfpr;ir mspf;f \$lhJ”

- gpshl;Nlh

“kdeyk; kd;Daph; Mf;fk; - ,dey;
vy;yhg; GfOk; jUk;”

- jpUts;Sth;

Thereby patients were advised to do “**Yoga Practice**”i.e Pranayamam (gpuzhahkk;).
Several hundred years before,Siddhar Thirumoolar mentioned the importance of

“**Pranayamam**” by the following lines.

“ Gwg;gl;L Gf;Fj; jphfpd;w thAit
 newpg;gl cs;Ns epd; kykhf;fpy;
 cWg;G rptf;Fk;> cNuhkk; fUf;Fk;
 Gwg;gl;Lg; Nghfh; GhprilNahNd”
 -jpU%yh;

ii) “Asanas”(Mrdq;fs;):

These asanas relieve patient’s strain and stress and also prevent the Kalanchagapadai disease. Patients were advised to do the following asanas.

Padhmasana	(gj;khrdk);
Sarvangasana	(rh;thq;fhrdk;)
Shavasana	(rthr;dk;)
Garudasana	(fUlh;rdk;)
Gomukasana	(NfhKfhrdk;)
Yogamuthra	(Nahf K; jpiu)

gj;khrdk;:

rkjsj;jpy; rk;kzkpl;L cl;fhh;e;J tyg;ghjj;ij ,lj;njhil kPJk;> ,lg;ghjj;ij tyJ njhil kPJk; Vw;wp
 ,uz;L iffisAk; Kd;Gwk; xd;wk; kPJ xd;whf kyh;e;jpUf;FkhW ,Uj;jy;.
 cly;eyKk>; kd kfpo;r;rpAk; Vw;gLk;.

rh;thq;fhrdk;:

ky;yhe;J gLj;J fhy;fis nkJthf xl;ba gbNa NkNY Jhf;fpg; gpd; Gl;l ghfj;ijAk;>
 ,Lg;Gg; ghfj;ijAk; NkNy Jhf;fpf; iffshy; KJFg; Gwj;jpy; jhq;fp epw;wy;.

“tPjd Nfhsk; Jhz;lg;gLfpwJ.

eiu>jpiu> %g;G khwp ,sikAz;lhFk;

clypd; vy;yh cWg;GfSk; gyg;gLk;

G+uz rtrhe;jp Mrdk;:

ky;yhe;J gLj;Jf; fhy;fis NeuAk; iffis clNyhL gf;fthl;bYk; itj;J NeuHfg; gLj;J ,Uj;jy;.

,J fisg;igg; Nghf;Fk;. Gj;Jzh;r;rpia cz;lhf;Fk;

fUlhrdk;:

Neuhf epw;fTk;. tyJ fhy; NeuHf itf;f Ntz;Lk;. ,IJ njhilia tyJ njhilNky; itj;J ,IJ fhYk;> ghjKk; tyJ nfz;ilf;fhiyr; Rw;wp ,Uf;fNtz;Lk;. tyJ fhyhy; epw;fTk;. iffisAk; mNjNghy; khh;Gf;F Kd; klf;fp tyJ ifapy; ,IJ ifiar; Rw;wp ,U es;sq;iffSk; njhLkhW Kfj;jpw;F NeuHf itf;fTk;. fz;fs; %ba epiyapy; ,Uf;fTk;.

fz;ghh;it kpFk;

iffhy; %l;Lfs; gyg;gLk;

kdk; xLq;Fk;> mwpT NkNyhq;Fk;.

NfhKfhrdk;:

,uz;L fhy;fisAk; ePl;b cl;fhuTk;.,IJ fhiy klf;fp tyJ fhYf;F fPo; itj;J ,IJ Fjpfhy; tyJ gpl;l;jpy; gLkhW itf;fTk;. tyJ fhiy klf;fp ,IJ fhYf;F Nky; itj;J Fjpfhy; ,IJ gpl;l;jpy; gLkhW itf;fTk;. ,uz;L Koq;fhy;fSk; xd;wd; Nky; xd;whf xNu NeuHf ,Uf;FkhW nra;aTk;. iffis Koq;fhy; Nky; xd;wd; Nky; xd;whf itf;fyhk; my;yJ gf;fthl;by; ,uz;L ghjq;fisAk; gpbj;Jf; nfhs;syhk;. fz;fs; %ba epiyapy; ,Uf;f Ntz;Lk;.

,J ,sikia cz;lhf;fp> tNahjpfj;ij khw;WfpwJ.

Jhf;fkpd;ikia ePf;FfpwJ. kd ,Wf;fk; ePq;FfpwJ.

Nahf Kj;uh:

gj;khrdj;jpy; mkuTk;> ,uz;L iffisAk; gpd;Gwk; vLj;Jr; nrd;W fl;bf; nfhs;sTk;>
fz;fis %lTk;. nkJthf Kd;Gwk; clk;ig tisj;J new;wp jiuapy; njhLkhW nra;aTk;.
tapw;Wf; NfhshWfs; ePq;Fk;.
kyr;rpf;fy; epth;j;jpailAk;
kdk; mikjp ngWk;.
KJF jz;Ltl typ> jiytyp> tapw;Wtyp> FzkhFk;.

gj;khrdk;



rh;thq;fhrdk;



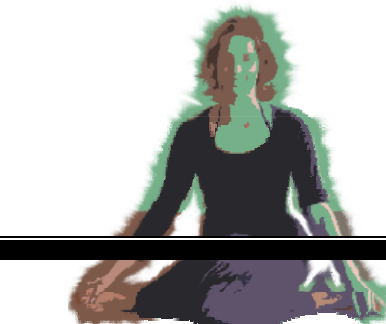
rthrdk;



fUlhrrdk;



NfhKfhrdk;



Nahf Kj;uh



7. ,sntapypy; fha;jy; (Sun therapy) ie. “#hpa ek];fhuk;”

“khiy ntapw; Fzk;”

“khiy ntapw; fha;e;jhh;FW gpzp Kw;fhrW kpyf;fzEhy;

Ma;e;jhh;f; FWtOr; nrhyhk;.”

-khiy ntapypy; fha;e;jth;fl;F gpzpf; >

goikahfpa Fw;wkw;w ,yf;fz Ehw;fis Muha;r;rp nra;j Gyth;fl;Ff; fhl;rpapy; gl;l tOr;
nrhw;fs; ePq;FtJ Nghy ePq;fptpLk;.

8. Advised to take “Rejuvenation”(fhafy;gk;) to render the body invulnerable.

All patients were also advised to follow siddhars preventive measures which would give immortality of body and soul,quoted in “Pathartha Guna Chinthamani” as follows.

“jpz;z kpuz;Ls;Ns rpf;f tlf;fhkw;

ngz;zpd;gh nyhd;iwg; ngUf;fhky;-cz;Zq;fhy;

ePh;RUf;fp Nkhh; ngUf;fp> nea;AUf;fp Az;gth;jk;

NgUiuf;fpw; NghNk gpzp”

“ghYZ;Nghk;;: vz;nza;ngwpd; nte;ePhpw; Fspg;Nghk;;

gfw;GzNuhk;;: gfw;WapNyhk;;: gNahjuK %j;j

VyQ;Nrh; FoypaNuh bsntapYk; tpUk;Nghk;

,uz;llf;Nfhk;;: xd;iw tpNlhk;;: ,lJ ifapw; gLg;Nghk;

%yQ;Nrh; fwp EfNuhk;> %j;j japh; cz;Nghk;.

Kjdhspw; rikj;jfwp aKnjdpD kUe;Njhk;

Qhye;jhd; te;jpbDk; grpj;njhopa Tz;Nzhk;
ekdhh;f;fpq; NfJfit ehkpUf;F kplj;Nj.”

cz;gjpU nghOnjhopa %d;W nghOJz;Nzhk;
cwq;Ftnjjp uhnthopag; gfYwf;fQ; nra;Nahk;
ngz; flikj; jpq;fSf;Nfhh; fhyd;wp kUNthk;
ngUe;jhf nkLj;jpbDk; ngah;j;J – ePuUe;Njhk;
kz; guT fpoq;Ffspw; fUizapd;wp GrpNahk;
thioapsk; gpQ;nrhopaf; fdpapUj;jy; nra;Nahk;.
ez;Gngw Tz;lcpd;G FWeilAq; nfhs;Nthk;
ekdhh;f;fpq; NfJit ehkpUf;Fkplj;Nj.”

MW jpq;fl; nfhU jlit tkd kUe; japy;Nthk;
mlh; ehd;F kjpf;nfhUfhw; NgjpAiw Efh;Nthk;
NjWkjp nahd;wiuf;Nfhh; juerpak; ngWNthk;
jpq;fsiuf; fpuz;LjuQ; rtstpUg; GUNthk;
tPWjrh; ehlnfhUfhy; nea; KOf;if jtpNuhk;
tpopfSf;fQ; rd%d;W ehl;nfhUfh ypLNthk;
ehWfe;jk; Gl;gkpit eLeprpapd; KfNuhk;
ekdhh;f;fpq; NfJfit ehkpUf;F kplj;Nj”.
gfj;njhOf;F khjurq; fue;Jiulg;g kpit e;Jhl;
glneUq;Nfhk;: jPg ike;jh; kuepoypy; trpNahk;
Rfg;Gzh;r;rp ardgr dj;jUzQ; nra;Nahk;.
JQ;rYz tpUkyQ;ir NahkOf; fhil
tFg;ngLf;fpw; rpe;Jfr kpit khiy tpUk;Nghk;
tw;rye;nja; tk;gpJh;rw; FUit tpl khl;Nlhk;
efr;ryK Kistr;ryKe; njwpf;Fkpl kZNFhk;
ekdhh;f;fpq; NfJfit ehkpUf;F kplj;Nj”

-gjhj;j Fz rpe;jhkzp

Diet(czT) and Habits(gof;ftof;fq;fs;):

“xU ehl;ilf; fhty; nra;Ak; Nritapy; kd typikiaf; fhty; nra;tJ czT.”

- nky;nyz;lh;

“ khWghby;yhj cz;b kWj;Jz;zpy;

CWghby;iy caph;f;F”

- jpUts;Sth;

According to siddha system “Kalanchaga Padai” and other skin ailments are due to the “Vitiation of Vatha”.So brings down to vitiated vadha,the patients were advised to follow the following food regimens.

“thj Nehia ePfFk; nghUl;fs;”:

“nrq;fOePh; Nfh\;le; Njd;kpsF ey;nyz;nza;

jq;FngUq;fhae; jhOjhio – vq;nfq;Fk;

\$l;LrpW Kj;Jnea; Nfhjpy; cSe;jpitfs;

thl;Lkdp yj;ij kjp”

- gjhh;j;j Fz rpe;jhkzp

czTld; Nrh;f;f;\$ba nghUl;fs;:

nra;fOePh; fpoq;F - ey;nyz;nza;

Nfh\;lk; - ngUq;fhak;

Njd; - jOjhio

kpsF - Mkzf;F nea;

- cSe;J

2. gj;jpa gjhh;j;jq;fs;

nfhs;S fhb Fkl;bf;fha;
Nfhop gd;wp nfhf;FINd
Ks;spw; nghpa ghfw;fha;
KjpUk; mtiu gaw;whq;fha;
gs;sj; njOe;j klw;NrK;G
glUk; ts;sp ghiyapit
vs;sj; jidjhd; jp;d;gPNuy;
vy;yh kUe;Jk; ,oe;jPNu
-gjhh;j;j Fz rpe;jhkzp

nfhs;S
fhb
Fkl;bf;fha;
Nfhop
gd;wp
nfhf;F
nghpa ghfw;fha;
Kw;wpa mtiuf;fha;
gaw;wq;fha;
NrK;Gf; fpoq;F
ts;spf;fpoq;F

3.Avoid Karappan food items.

“ ngUQ; Nrhs kWfK; ngUk; fk;G
tuF fhUld; thioapd; fhnaHL
ciunfhs; ghfw;nfhspw;W kPd; cz;bby;

tphtjha;f fug;ghD kpFe;jNj”

gjhh;j;j Fz rpe;jhkzp

4. “GspJth; tpQ;R fwpahw; G+hpF;Fk; thjk;”

gjhh;j;j Fz rpe;jhkzp

5.Avoid guava,egg,fish,chicken.

6.Avoid alcohol,smoking etc.,

7.Should not be stressed and strained.

8.Since Kalanchaga Padai is a chronic disorder,avoid heavy drugs.

9.Practice Yoga and administered meditation to calm the mind and body from stress and strain.

10.Patches in Kalanchaga Padai lesion should be washed with like warm for everyday and then applied external medication.

11.Provided nutritional diet to avoid nutritional problems including vitamin,mineral and protein.

12.Avoid high protein value diet to prevent keratinisation of the skin ,which will occur in Kalanchaga Padai.

13.Foods contain high protein value:

Grams and dal - 22gm/100gm

Meat and related products - 20gm/100gm

Nuts and oil seeds - 19gm/100gm

Wheat - 11gm/100gm

Food contain low protein value:

Roots and tubers - 1.3gm/100gm

Other vegetable	- 1.9gm/100gm
Fruits	- 8gm/100gm
Green leafy vegetables	- 4gm/100gm
Milk and curd (Cow's)	- 3.2gm/100gm
Raagi, rice and rice products	-7.0gm/100gm

Neekam(Treatment):

The aim of treatment is based on

- 1.To bring the three doshas to equilibrium.
- 2.To treat the patients according to symptoms,by internal medicine,"Paragichakkai Chooranam" as well as external medicine "Thaser meni Ennai".
- 3.To build up seven body constituents and increase natural immunity.
- 4.Diet.
- 5.Treatment for Karma of previous incarnation.

"Kg;gpzp kUtp KwpT nfhs; Fwpg;ig
jg;ghjwpAk; jd;ikAk; thjcpj;j itag;
gphpitAk;ik tjhk;"

"Vwpapwq;fp ,ize;J fye;J
khwp khwp tUQ; nra;ifaha; gpzp
Neh;ikawpe;J ePl;L kUe;Nj
rPhpajh nkdr; nrg;Gth; rpj;jNu"

For normalizing Thri dohas,

“tpNurdj;jhy; thjk; jhOk;”

tkdj;jhy; gpj;jk; jhOk;

erpa mQ;rdj;jhy; fgk; jhOk;”

“XJfpd;w kyf;fl;il nahopa itj;jhy;

clypYs;s thijnayhnhkLq;fpg; NghFk;.”

“mwpe;jpLk; thjk; mlq;F kyj;jpdpy;”

5gm of Nilavagai choornam with water at bed time (or) 15ml Vellai Ennai with hot water was administred at early morning as a “Kalicchal Medicine” before starting the treatment to bring out the vitiated vatha into normal.

- ✓ The patients were advised to use “Nalunguma” instead of soap.
- ✓ The Patients were also advised to wear fresh cotton clothes and to avoid cosmetics.
- ✓ Treatment for”Karma of previous birth”(incarnation).

Agasthiyar quoted as follows.

“ fd;kk; jPu”

“jaq;fhky; jPUtjw;F tifiap; NfS

jd;ikAs;s tU\kJ jifAq;fhyk;

eaq;fhk yhba khthirad;W

ed;ikAs;s njspePhpy;];ehdk; gz;zp

Kaq;fhkw; rptjyj;ijf; Nfhhp te;J

Kj;jpAl dhapuk; Ngh; Ntjpah;f;F
Rak;ghfj;jhd; nfhLj;J etf;fpfe; jd;idj;
Njhj;jhpj;J ml;rijAk; thq;fpf; nfhs;Ns.”

“thq;fpNa nghjpiff;Fj; njd;fpof;F %iy
thpiAs;s thdkh kiyf;Fr; nrd;why;
Jhq;fpNa ahypiyNkw; gs;sp nfhz;l
Rthkpapl re;epjpapy; jpahdQ; nra;J
Njq;fp NaAjf nkd;w fpzuq; Fz;L
jpukhf xU nrk;G nea;ia thNu.”

nra;As;-216

“thh;j;JNk kW nrk;G japynkhz;L
ike;jNd NtLfl;bf;nfhz;L Ngha; eP
Mw;WNk a;epjre;jp fhnrilaha;f; nfhs;s
mg;gNd jpq;fsJ %d;Wf;Fs;Ns
Njhw;WNk NjfkJ Nghd;NghNy jhd;
Jaukhd fd;kNdha; JhuNthLq;
fhj;JNk apj;japyq; nfhs;shtpl;lhy;
fd;kNeha; jPuhJapd;dq;fNs”

-nra;As; - 217

Since siddha system of medicine is based on the mukkutra theory, the treatment is mainly aimed to bring down the three dhoshas to its equilibrium state and thereby restoring the physiological condition of various thathus.

Anupanam is Siddha System

“mDghdj;jhNy atpo;jk; gypf;Fk;
,dpjhd Rf;F fd;dy; ,Q;rp – gpDKjfhhy;

NfhNkak; ghy; Kiyg;ghy; Nfhnea; Njd; ntw;wpiy ePh;
Mkpijahuha;e;J nra;ayhk;.”

- Njiuah; ntz;gh

Siddha system considers anupanam as an important and sometimes more important than the medicine itself. Without a knowledge of the importance of anupanam, success in the treatment is not possible.

Pathiyam

During the course of treatment, the drug is administered to the patients according to nature of the disease and the patients were advised to follow certain precautions regarding diet and physical activities.

This type of medical advice in Siddha system of medicing is termed as “Pathiyam”, importance of pathiyam is quoted as follows.

“gj;jpaj;jpdhNy gyDz;lhfK; kUe;J
gj;jpaq;fs; Nghdhy; gyd; NghFk; - gj;jpaj;jpy;
gj;jpaNk ntw;wp jUk; gz;bjh;f;F Mjypdhy;
gj;jpaNk cj;jpnad;W ghh;.”

-Njiuah; ntz;gh

Niraivu:Restoration

- 1.Reassurance of disease recovery was given to all patients.
- 2.All the patients were advised to live in good health free from diseases.

B. MODERN ASPECTS

SKIN ANATOMY & PHYSIOLOGY

The skin , the body`s largest magnificent , complex , multi purpose organ , covering the entire outside of the body and weighing approximately 4 kgs and has about 1.75 sq meters of surface area.

Throughout the body , the skin`s characteristics vary [that is thickness, colour , texture]. For instance , the head contains more hair follicles than anywhere else , while the soles of feet contain none In addition , the sole of the feet and palms of the hands have much thicker layers .

The skin is made up of the following layers . with each layer performing specific functions .

- Epidermis
- Dermis
- Fat layer (or) Hypodermis (or) subcutis

The Epidermis :-

The epidermis, which thickness varies from 0.4mm to 1.6 mm , is a important layer . the langerhans cells , responsible for the immunology of the skin, the melanocytes and tyrosinase enzyme , responsible for the production of melanin and colour , are located in the epidermis .

This is the layer of skin to which , cleansing , exfoliative or hydrating product is applied. The epidermis holds a large amount of water .

The epidermis consists of the following layers from below to up ward .

1. Basal cell layer
- 1 Squamous cell layer
- 2 Granular cell layer
- 3 Stratum lucidum
- 4 Horny cell layer

Basalcell layer

The basal cell layer is composed of three distinct cell, the keratinocytes, the melanocytes and the merkel cells .

a. Keratinocytes

The keratinocytes produce certain chemicals called cytokines that are involved in radiation.

b. Melanocytes

These cells produce melanin granules [melanosomes], which are transformed to adjacent keratinocytes. Melanin protects the skin from the ultra violet radiation.

c. Merkel cells

They are touch receptors. They are very scarce and are occasionally arranged in groups.

Squamous cell layer

It is also known as the prickly cell layer. These cells are polygonal in shape. This layer is 5-12 cell in thickness.

Langerhans cells are found in this layer. They are bone marrow derived cells. They function as the macrophages of the epidermis, processing contact antigens, which are presented to sensitized T-lymphocytes.

Granular cell layer

It is also as stratum granulosum. The cells of this layer are flattened and filled with keratohyaline granules.

Stratum lucidum

It is inner most portion of the stratum corneum. It is thin but best seen in the palms and soles where the horny layer is thickest.

Horny cell layer

It is the so called stratum corneum. It is characterized by strength by strength, flexibility, elasticity, toughness and a dry surface that discourages the growth of microorganisms. This layer is renewed every 28 days with basal cells migrating from the basal layer to the top surface. This is a dynamic and progressive process.

EPIDERMAL APPENDAGES

The following glands and structures are found in the epidermis:

1. Eccrine glands
2. Apocrine glands
3. Sebaceous gland
4. Hair follicles
5. The nails

1. Eccrine glands

These are the sweat glands. They are found in abundance throughout the skin surface, excepting of vermilion of the lip, labia minora, glans penis and inner aspect of the prepuce and are found in highest concentration on the palm, soles and the axillae. They secrete sweat that is a hypotonic solution, which permits evaporative cooling of the body.

2. Apocrine glands

These are the scent glands. They are found primarily in the axillae and anogenital region. They produce scent. Modified apocrine glands are found in the ear canal, eyelids and in the breasts.

3. Sebaceous glands

These are found on all parts of the body except the palms and soles. They produce oil that lubricates and protects the skin and hair. This oil is called the sebum.

4. Hair

Hair is found on almost every part of the body surface except on the palms, soles, the dorsal surface of the terminal phalanges. Hair differ in length, thickness and colour in different part of the body and in different races, there are three types of hair

1. Long , medullated, pigmented hair seen on the scalp
2. Short , fine , non- medullated “ lanugo“ hair seen in women,children and on the faces and trunks of adults [vellus hair]
- 3 Thick bristles seen in the nose and the ears .

Hair grows about 1-2 cm per month. The growth varies in different people races and also on the different parts of the body. Hair growth and development is under endocrine control .

5. Nails

These are semi – transparent , plate – like horny structures , covering the dorsal surfaces of the distal phalanges of the fingers and toes. The proximal edge of the nail is known as the nail root of the nail; the visible portion of the nail is called the nail plate. It is semi – transparent and looks red due to the abundant vascular supply in the nail bed. The more opaque and rather whitish semi – lunar portion of the nail plate near its root is known as the lunula .

The surface of the skin on which the nail rests is known as the nail bed .nails may be objects of admiration and beauty. The material of the true nail develops from the matrix.

The Dermis :-

The second layer or dermis which is 5-7 times thicker than the Epidermis, lies below the epidermis and is connected to it by the basement membrane .

The Dermis consists of a thick connective membrane criss-crossed by

- Blood vessels
- Lymphatic vessels
- Nerve fibers
- Many sensory nerve endings

Collagen and elastin protein fibers the two main components of the dermis, It act as a structural support system for the nerve fibers, hair follicles , blood vessels and oil and sweat glands located in this layer, and also provide the skin with strength and elasticity.

The dermis contains few cells which are fibro blasts, mast cells, histocytes (or) macrophages, Lymphocytes and melanocytes.

The Hypodermis :-

The Hypodermis is the skin is third and the last layer , connects the skin with the muscle tissues .

This layer is highly elastic and has fat cells acting as “ shock absorbers” thereby supporting delicate structures such as blood vessels and nerves .

Blood vessels :-

The blood supply of the skin originates from a large number of anterior forming anastomosis in the deepest part of the cortex. From here single vessels runm upward and form a second network in the upper cortex. Finally terminal arterioles ascent into the papillae ending in capillary loops which drain in to connecting venules. The blood is returned to the veins in the subcutaneous tissue.

Lymphatics :-

The skin contains a rich network of lymphatics, which drain into a few a larger vessels in the hypodermis.

Nerve supply :

The nerve supply of the skin consists of a motor sympathetic portion derived from the sympathetic portion derived from the sympathetic ganglia and sensory spinal portion arising from the dorsal root ganglia. The sympathetic fibres innervate the blood vessels , erector pilorum muscles and apocrine duct , where the fibers are adrenergic and cause contraction.

PHYSIOLOGY :

1. Protective function:

The epidermis and subcutaneous fat play roles in the protective functions, the mechanical properties of the skin depends mainly on the dermis. It protects the penetration of harmful substances and bacterial invasions. Another is to protect against sunlight by synthesis of melanin pigment.

2. Immunological function

The skin is in the front line of the defences of the body. In essence the defence involves, the protection of antibody- complex, multi hair proteins which bind with the offensive antigens, Langerhans cells probably play a crucial role in the contact sensitization, Immune surveillance against viral infections and neoplasms.

3. Sensory functions:

The skin is richly supplied with nerves and various types of specialized sensory end-organs, which provide information regarding environmental changes, so that the body can then adjust its activities accordingly. In some animals, the hair at certain situations have specialized sensory receptors located at the basis of the hair follicles which serve to enhance sensory appreciation

4. Secretion and Excretion:

The skin possesses various types of glands, which pour secretions on the surface. The more important glands are sweat and sebaceous glands. The eccrine glands which are scattered all over the body surface secrete a thin, transparent, watery fluid known as true sweat which the apocrine glands secrete a thicker rather milky and odoriferous solution.

Sweat in its composition consists of 1.2% solids and 98.8% water. The substances excreted in it are sodium chloride, sodium phosphate, sodium bicarbonate, keratin and a small amount of urea. The skin can also excrete certain drugs administered to the individual for example mercury, arsenic, iodine etc.

The sebaceous glands of the skin secrete sebums, which is composed of fatty acids, cholesterol, alcohol etc. Fatty acids which have a mild fungistatic activity. The sebum acts as a lubricant for the drying effects of atmosphere.

5. Synthesis of Vitamin D:

Vitamin D is synthesized in the skin as a result of exposure to ultra violet 'B' (UVB) radiation and, since it is carried in the blood attached to a binding protein to exercise a specific effect at a different site. Vitamin D₅ is essential for skeletal development, and it contains antirachitic properties. Vitamin D₃ is formed principally in the stratum spinosum and the stratum from the precursor 7-dehydrocholesterol by way of a previtamin D₃ (2,5).

6. Body heat regulation:

The skin plays a most important role in regulation of heat loss. It loses heat to the external environment in three ways by conduction, by radiation and by evaporation. Heat loss by the first two mechanisms takes place when the environmental temperature is lower than that of the skin. Heat loss by

evaporation mainly means the amount of heat spent by the body to evaporate the sweat from the surface of the skin. About 90% of the total heat loss of the body is regulated by the skin.

The heat loss through the skin is regulated by various

physiological mechanism. Which includes,

1. The reaction of the cutaneous vessels.
2. The reaction of the smooth muscle fibres of the skin and
3. Perspiration.

7. Endocrine function:

Hair follicles and sebaceous glands are the targets for androgenic steroids secreted by the gonads and the adrenal cortex and melanocytes and directly influenced by polypeptide hormones of the pituitary.

8. Storage function of skin

Blood is stored in the rich sub papillary plexus of the dermis, about one litre. The skin is also a good store house of ergosterol is irradiated by the ultra violet light of the sun and converted into Vitamin D.

The junction between dermis and hypodermis has a considerable capacity for storing fat and permanent store of subcutaneous adipose tissue. Certain substances like glucose and chloride also acts as a reservoir for topically applied corticosteroids (or) other hormones. Which absorbed slowly for many days from the skin surface.

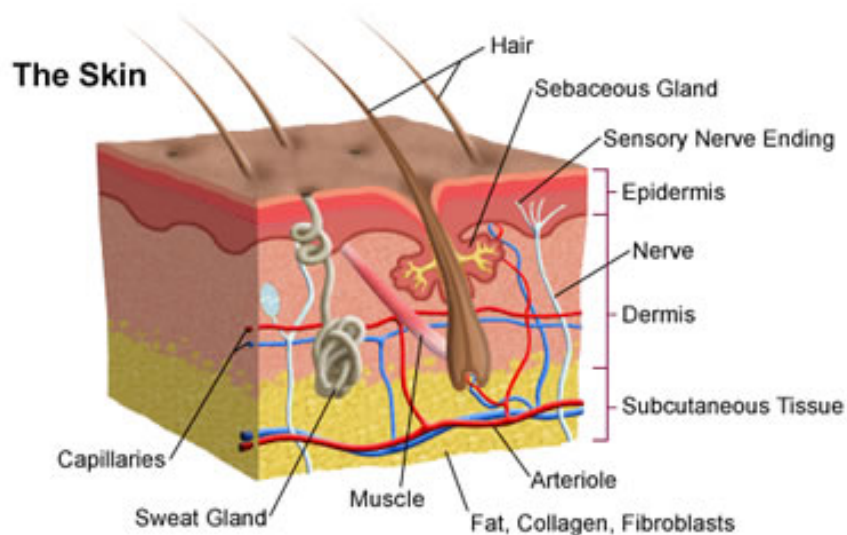
9. Absorption:

The skin can absorb substances dissolved in fatty solvents like vitamin and hormones.

10. Gaseous exchange through skin:

A small amount of gaseous exchange occurs through the skin. In man the amount of CO_2 exchanged through the skin is negligible compared to the amount exhaled from the lungs.

ANATOMY OF THE SKIN

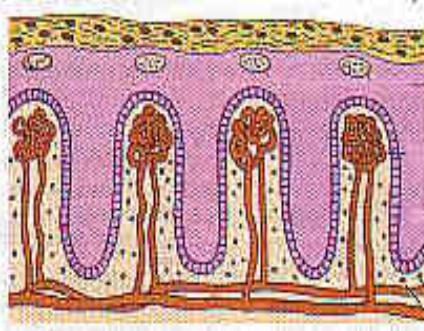


HISTOLOGY OF THE SKIN

NORMAL SKIN



PSORIATIC SKIN



PSORIASIS

Definition of Psoriasis : Psoriasis is a non-infectious, chronic inflammatory disease of the skin, characterized by well defined erythematous plaques with silvery scales which have predilection for the extensor surfaces and scalp, and by a chronic fluctuating course.

Causes of Psoriasis:

Psoriasis and immune system are closely linked. Psoriasis occurs when immune system (natural protection against bacteria, viruses, and other foreign invaders) does not work properly, and starts changing the behaviour of the skin cells. No one knows what triggers this.

A small group of scientists believe that **“Bacteria and fungi may activate psoriasis”**.

“Heredity” is likely to have some role in this condition. There is an increased prevalence of HLA Cw6. If it has one parent with psoriasis, it has a 25% chance of having it too. If both parents have it, chances are 50%; develop psoriasis earlier, and with more severe symptoms.

But some people with no family history have severe psoriasis, too. For this reason, it is believed that a **“combination of factors”** may cause the disease.

Precipitating factors:

Trauma :

When the condition erupting lesions appear in areas of skin damage such as scratches or surgical wounds (Koebner Phenomenon)

Infection :

β -haemolytic streptococcal throat infections often precede guttate psoriasis.

Sunlight:

Rarely, ultraviolet radiation may worsen psoriasis.

Drugs:

Anti-malarials, β -blockers and lithium may worsen psoriasis and rash may “rebound” after stopping systemic corticosteroids or potent local corticosteroids.

Emotion:

Anxiety precipitates some exacerbations.

Epidemiology and Incidence:

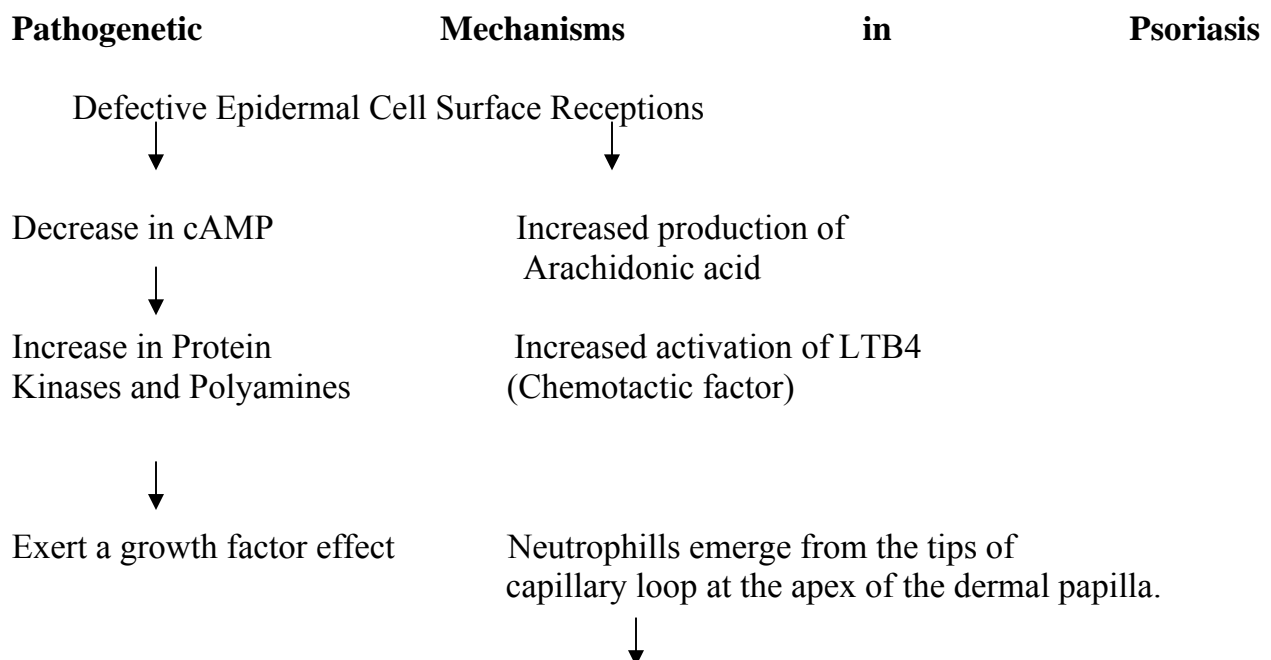
Psoriasis is found all over the world lower incidence in people of African origin, some Asian communities such as Japanese.

Research indicates that between 4.5 and nearly 7 million people in the U.S. have Psoriasis. It affects men and women equal rates and affects all age groups. On average, Psoriasis begins between ages 15 and 35, but it can begin at any time. About 10% - 15% of people with Psoriasis develop the condition before they're 10 years old.

Although about 80% of people with psoriasis have Plaque Psoriasis. Patients with AIDS may also develop very extensive psoriasis, that is resistant to current therapies.

Infection, especially with haemolytic streptococci, is not uncommon inciting influence in children. (Guttate form of psoriasis). Psoriasis has also appeared after bacterial and certain viral infections and has cleared with resolution of the infection.

Pregnancy may exert a salutary influence on psoriasis, possibly as a result of increased corticosteroid secretion. Physicians who have followed large numbers of patients with psoriasis provocative, influence of emotional stress in this disease.

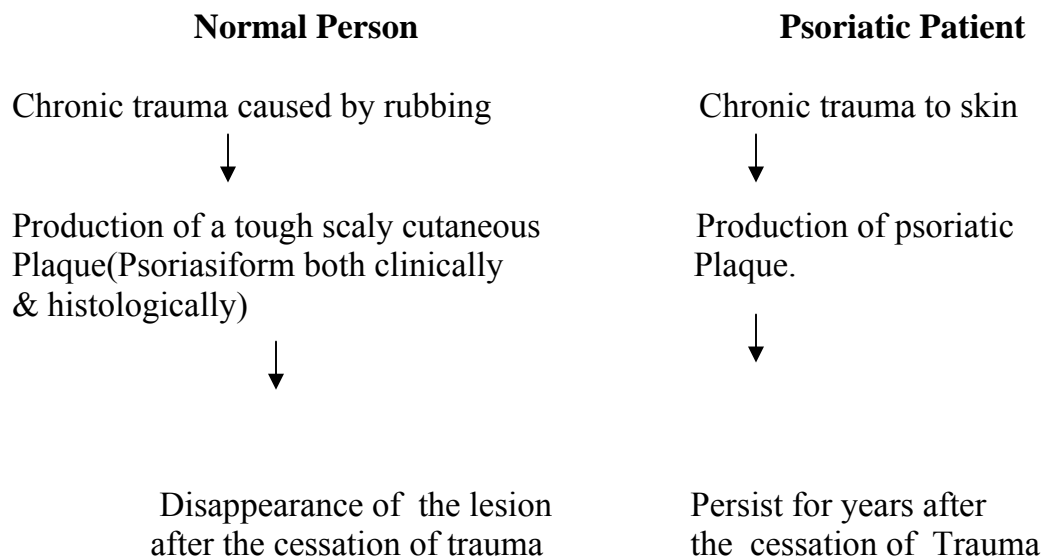


Aggregated neutrophils in the stratum comeum known as Monro microabscess.

Special areas of pathogenetic importance

Pathogenesis of Psoriasis according to Rubin & Farber:

The Pathogenesis of psoriatic plaques are appreciated by comparing the effect of chronic cutaneous trauma in persons with and without psoriasis.



Explanation for this unique response to injury:

- (1). After epidermal growth.
- (2). Abnormality in the microcirculation of dermis.

This abnormal epidermal proliferation related to defective epidermal cell surface receptors.

The capillary loops of dermal papillae become venular. This vascular change causes extrusion(Squirting) of many neutrophils at the tips of the dermal papillae, after which the neutrophils migrate through the epidermis. The dense collections of neutrophils in stratum corneum is called mono micro abscesses.

Special areas of pathogenetic importance

Recent investigation & data emphasize the complexity of the disease process and broaden our understanding of the clinical features, course and treatment of the disease. Accordingly, a discussion of the major findings including epidermal kinetics, immunologic alterations, polymorphonuclear leucocyte activity, vascular abnormalities and viral agents, is appropriate. The interrelation of these influences is only partially understood, and their pathogenetic importance is, still speculative. Generally accepted fundamental tenets relating to psoriasis pathogenesis, are:

1. Accelerated proliferation of keratinocytes and disturbed epidermal maturation are primary alterations in psoriasis.

2. Essential clinical features of pathogenetic significance include the hereditary nature of psoriasis, a course marked by spontaneous exacerbations and remissions and the predilection of lesions to certain sites.

3. The biochemical alterations of psoriasis include

- (a) increased DNA replication
- (b) altered cyclic nucleotide levels,
- (c) abnormalities in prostaglandins and their precursors, and
- (d) altered carbohydrate metabolism.

(i) Epidermal kinetics

The cardinal patho-physiologic event in psoriasis is an increase in epidermal cell proliferation, (measured using Glycine 14.C). Expansion of the germinative cell proliferation in psoriasis

is the result of mitotic activity in three layers of cells in the basal zone, as compared with one layer of basal cells in uninvolved skin. Moreover, the length of these layers are

extended over three-fold by the enlarged dermal papillae in psoriasis. Psoriatic epidermis turns over 7 times faster than normal skin. The transit time of cells through the stratum malpighii is 2 days in psoriatic epidermis and 13 days in normal epidermis. Although there is debate regarding the exact values, it is clear that the turnover time in psoriatic epidermis is significantly decreased.

(ii) Polymorphonuclear leucocytes:

Polymorphonuclear leucocytes play an important role in the pathogenesis of psoriasis. PMNL's migrate into the epidermis, and their focal accumulation in the stratum corneum, known as Monro micro abscesses, is a consistent histologic finding.

(iii) Vascular abnormalities:

The dermal capillary loops of both involved and uninvolved skin of psoriatic patients are dilated and abnormally tortuous. Neutrophils and enzyme may be "Squirted" into the epidermis from these distorted vessels and stimulate some of the earliest dermo – epidermal changes. Electron microscopic studies have revealed markedly attenuated vessel walls and gaps between the endothelial cells.

(iv) Immunopathology:

There is some evidence pointing to defects in cellular immunity in psoriatic patients. Decrease in E-rosette-forming T lymphocytes in psoriatic patients is associated with the active phase of the disease and that it disappeared in 4 to 6 weeks after the onset of remission. This suggested that the reduction in T cells is transitional.

7. Viral agents:

Over the years, infectious agents of all types have been proposed as the possible cause of psoriasis. Recently, the possible etiologic role of viruses has resurfaced with the finding of virus-like particles in the skin and in PHA stimulated lymphocyte cultures from psoriatic patients. T cell defects in psoriasis were thus described to such viral infection. Hellgren et al. also demonstrated virus-like particles in sedimented material of

ten patients with severe psoriasis and 3 patients with mild disease, which were not found in 20 healthy controls.

Viral activation under proper environmental conditions, it is suggested could be responsible for the cell proliferation, abnormal pharmacology, and autoimmune phenomena in psoriasis.

Histo-Pathology :

The psoriatic lesion is composed of 2 distinct morphological changes:

- (1) The dermal capillaries are markedly elongated and coiled with irregular fusiform dilatations of their walls.
- (2) Relating to epidermis.

Normally it takes 27 days to replace the epidermis (i.e. from basal cell to fully keratinized horny cells). In psoriasis this takes only 3-4 days. As a result the horny layer is immature and parakeratotic with nuclear fragments still present in the horny cells. (This parakeratosis is responsible for the silvery scaling, a characteristic of psoriatic lesion).

The microscopic features of psoriasis reflect

- (a) The increased cellular activity of the epidermis
- (b) The dermal alterations.

Then show some variation, however, depending on the age and clinical form of the lesions, their location and the influence of treatment.

A study of the earliest visible lesions, called "Prepoint papules" by Chowanec et al, revealed a predominantly neutrophilic infiltrate intravascularly in the dermis and invasion by neutrophils of an edematous epidermis.

The salient histopathologic findings of psoriasis include.

- (1) Acanthosis, with elongation of the rete ridges.

- (2) Elongation and clubbing of the dermal papillae.
- (3) Parakeratosis, usually dominating an associated hyperkeratosis.
- (4) Thinning or absence of the granular layer, a consequence of the epidermal proliferation.
- (5) Increased mitosis primarily in the multiple layers of basal cells of the psoriatic lesions and possibly in the lower malpighian layer. This is to be contrasted with the presence of mitosis only in the single basal cell layer of normal skin.
- (6) Edema and a mild to moderate dermal infiltrate consisting primarily of lymphocytes and monocytes.
- (7) Epidermal microabscesses (Monro micro abscesses) composed of focal collections neutrophils in the stratum corneum or immediately below it.

Clinical discussion

Psoriasis presents many faces to the clinician. Although its ravages are limited essentially to the skin and joints, it is remarkably a protein disorder which produces a surprising variety of clinical patterns.

Distortions of the nails are also frequent findings in the psoriatic, many times clinching the clinical diagnosis. Alopecia or even minor disturbances in the quality of hair or its rate of growth are never seen. The clinical discussion here begins with (1) a description of the vital qualities of the psoriatic skin lesion and (2) continues with a consideration of the sites of predilection, (3) the Koebner's phenomenon, (4) the

bacteriology of psoriatic skin (5) psoriatic nail disease(6) the various clinical forms of psoriasis,(7) psoriatic arthritis and (8) the systemic associations of psoriasis.

(A)Morphology of the psoriatic skin lesion:

A sharp,definable border, a bright red colour,and a silvery-white scales delineate the lesions of psoriasis.

The sharp border,which can usually be felt as well as seen,abruptly demarcates the epidermal hyperplasia and dermal changes of psoriasis.The skin immediately surrounding the psoriatic patch may appear pallor than normal skin,and is apparently less reactive to certain therapeutic and pharmacologic stimuli,possibly because of impending or resolving involvement by the inflammatory process.

The bright red colour,is indicative of the dilated superficial vasculature,particulary of the dermal papillae of psoriasis.These capillaries so closely approach the skin surface at the apex of the elongated dermal papillae that the removal of psoriatic scales frequently produces fine bleeding points(Auspitz sign).

The scale of psoriasis is an almost constant feature.Characteristically silvery white,scales become thicker.These scales are normally rather loosely adherent and are the result of the greatly accelerated and incomplete keratinization process.They may be very thin,usually curling slightly as they detach,or may be piled up and thickened to produce a kertain plate over the erythematous skin lesions.

Pustules are not a regular feature of most clinical types of psoriasis,like the lesions of pustular psoriasis,they apparently represent occasional enlargement, to a grossly visible size, of the microabcesses which are a cardinal miscrospic component of the diease.

Bullae have been seen during acute phases of psoriasis, but they are exceptionally rare developments of the pathologic process. Psoriasis does not produce scarring.

(b) Sites of Predilection:

It is important to realise that psoriasis does not produce the destructive cicatricial change of lichen planus, which may result in a band like pterygium or loss of the nail. Acute fulminant psoriasis or pustular psoriasis affecting the paronychia and proximal nail fold skin may also result in nail dystrophy. Common sites of predilection in the following order are:

- (1) Scalp
- (2) Back of Elbows
- (3) Front of Knees
- (4) Lumbo sacral region
- (5) Nails, palms and soles
- (6) Rarely Mucous membranes

The Koebner or Isomorphic phenomenon

Psoriatic skin lesions may first appear at sites of local injury, scars or vaccination. This phenomenon is known as Koebner's phenomenon. Although best known in psoriasis, the Koebner's phenomenon may also occur in certain other skin diseases, notably lichen planus, lichen nitidus, pityriasis rubra pilaris and Darier's disease. Moreover, a pathogenetic role for the Koebner's phenomenon in psoriatic arthritis has also been suggested. The important features of the Koebner's or isomorphic phenomenon in psoriasis can be summarized as follows:

1. The reaction may follow simple irritation, physical injury, wounds sunburn, x-ray radiation or vaccination or may occur in pre-existing disease, such as an eczematous dermatitis or an old scar.
 2. The trauma must reach or act on the papillary layer of the dermis, but epidermal injury is also necessary. Simple vasodilatation, vasoconstriction, or suction injury that spares the epidermis will not evoke the reaction.
 3. It occurs in almost half, but apparently not in all, psoriatics at some time during their disease.
 4. The Koebner reaction can be enhanced or inhibited by certain chemicals and cytotoxic agents.
 5. After the injury, an interval of 8 to 10 days usually (Range 3-18 days) precedes the development of the psoriatic skin lesion. Capillary alterations precede the skin changes.
 6. When it follows an accident, industrial injury, or medical therapy, the Koebner's phenomenon may have medicolegal importance.
 7. The practical significance of the Koebner's phenomenon as a precipitating cause of psoriatic skin lesions should be emphasized to all patients with the disease.
- Avoidance of local skin injury may significantly decrease the cutaneous involvement in some patients.

Bacteriology of Psoriasis

The colonization of psoriatic skin by bacteria results in :

Secondary infection & aggravation of the skin lesions.

(a) A significant source of infection to others.

Microbiologic studies have revealed increased number of staphylococcus aureus and resident cocci on the involved as opposed to the uninvolved skin of psoriatics, although the number of organisms found was not remarkably high. Moreover, occlusion of the skin

lesions with a vapour-impermeable plastic film led to dramatic increase of all organisms, especially *S. aureus*, which apparently increases disproportionately to the remainder of the flora.

Frank pustule formation or impetiginous crusting was not seen with this increased staphylococcal growth, but an intensification of the inflammatory process was evident, along with a relative resistance of the lesions to otherwise effective topical medicaments, such as corticosteroids. This refractoriness could be reversed by local treatment with broad-spectrum antibiotics. It is possible that the improvement in psoriatic skin lesions sometimes seen after systemic and local antibiotics may be directly attributable to the reduction of the microflora. Exfoliation of psoriasis scales is a definite hazard as a source of hospital psoriasis, both the localized and the generalized varieties, are sterile, at least until ruptured.

(e) Psoriatic Nail Diseases:

Nail involvement is common in psoriasis. Nails are involved in about 50% of cases. Finger nails show changes more often than the toe nails.

Nails show 3 types of lesions:

- (a) They may show multiple small pits like those on a thimble.
- (b) They may become detached from its bed over the distal half of the affected portion being opaque friable and discoloured.
- (c) They may become thickened rough and pigmented.

It is important to realize that psoriasis does not produce the destructive cicatricial change of lichen planus, which may result in a band like pterygium or loss of the nail.

Acute fulminant psoriasis or pustular psoriasis affecting the paronychia and proximal nail fold skin may also result in nail dystrophy.

Classic psoriatic arthritis affecting the distal interphalangeal joints is usually associated with nail dystrophy. Nail involvement in the absence of psoriatic skin lesions is rare and may be difficult to prove.

The thickened or onycholytic psoriatic nail or subungal tissue frequently contains *Candida albicans* and *Pseudomonas* organisms. The latter may at times produce a greenish discolouration of the nails. The psoriatic nail is, however, peculiarly resistant to invasion by superficial fungi.

Various Clinical forms of psoriasis

Common plaque or Nummular Psoriasis

The commonest form of the disease is the plaque or nummular variety in which round or oval lesions of one to many centimeters are found on the elbows, knees, scalp and trunk in insidious onset. This type of psoriasis may remain stationary for long periods of time. Exacerbations may bring small guttate lesions along with the large plaques. Serpiginous, annular, gyrate and zonal arrangement of lesions may be seen along with Koebner-induced linear papules produced by scratching. Thick inveterate plaques may occur in patients with very chronic involvement of the elbows, knees or hips.

Guttate Psoriasis

The abrupt appearance of a shower of small, drop-like psoriasis lesions with a less prominent scale than usual over much of the skin surface, but especially on the trunk and proximal extremities, should suggest this special syndrome. It is seen primarily in children and young adults and commonly occurs a few weeks after streptococcal infections.

Elevated antistreptolysin titers are usually found. The lesions often gradually disappear after resolution of the infection.

Palmar Psoriasis

A distinctive, patchy, hyperkeratotic type of psoriasis principally affecting contact points of the volar surface of the fingers and palms, this form of the disease may exist alone or in combination with mild psoriasis, usually of the elbows, knees and scalp. The lesions are related to local physical or chemical injury and are thus called Koebner phenomena. It may involve one palm more than the other as in tennis players, golfers or industrial workers, strangely, the same changes are less common on the soles. Palmar psoriasis is often confused with chronic contact dermatitis. It runs a protracted course of a few to several years, but may persist indefinitely. Palmar psoriasis is to be distinguished from pustular psoriasis of the palms and soles.

Psoriasiform (ITCH) neurodermatitis

Chronic psoriatic plaques may intensely provoke considerable scratching and a lichenoid appearance.

A rather common syndrome, however, is the lichenoid neurodermatitis of the occipital scalp and nape of the neck, which develops a psoriasiform appearance. The majority of patients with this condition, usually women, have no psoriasis elsewhere, and would best be regarded as having a primary neurodermatitis rather than psoriasis.

Pustular Psoriasis

There are two clinically dissimilar conditions to which the name pustular psoriasis has been applied, namely:

- (a) Localised pustular psoriasis.
- (b) Generalised pustular psoriasis

Localised Pustular Psoriasis

Also known as pustular psoriasis of the palms and soles, this sterile pustular eruption affects the digits and volar skin of the hands or feet, or both usually in the presence of papulosquamous psoriasis elsewhere.

In its commonest form, superficial lakes of pus appear in crops on a background of erythema, most commonly on the thenar or hypothenar eminences or center or inner aspect of the sole.

The individual pustules dry up, turn brown and exfoliate off, but the whole condition is very chronic and lasts years or decades.

Generalised pustular psoriasis

Pustular may become widespread and associated with sheets of vivid erythema. This is generalized pustular psoriasis. This is the severest form of psoriasis and is fatal in about a quarter of cases.

Flexural Psoriasis

This type of psoriasis occurs mostly in the body folds – such as groins, axillae and infra-mammary regions. Seen in middle-aged obese individuals, women rather than men. The lesions are well demarcated, with reduced scaling, frequently itch.

Psoriasis and Bullous pemphigoid

The co-existence of psoriasis and bullous pemphigoid has been described in at least 28 cases in the literature. In many of these patients the bullous pemphigoid was believed to have been precipitated by treatment of the psoriasis with topical anthralin, crude coal tar, salicylic acid, ultraviolet light (UVL), sunlight or PUVA.

Psoriasis arthropathica

In this form, psoriasis is associated with a type of erosive arthritis, very similar to Rheumatoid arthritis.

Except that :

- (a) Distal interphalangeal joints were affected in more than 50% of cases.
- (b) Rose – Waaler test negative in over 90% of cases.

The nails were also involved in over 90% as compared to the usual average of 50%. Joints of fingers, feet, ankles, knees and sacro-iliac are selectively affected; these joints swollen and painful. The psoriatic eruption & involvement of the joints may increase or decrease simultaneously.

Psoriatic arthritis

Although the association between psoriasis and an inflammatory arthritis has been known for over 150 years, the recognition of psoriatic arthritis as a distinct clinical entity awaited the accumulation of epidemiologic, genetic, clinical, radiologic and laboratory evidence over the past three decades.

Epidemic evidence

To prove that psoriatic arthritis is a distinct clinical entity:

Psoriasis has been shown to be more common among arthritis, and arthritics more common in psoriatics than either disease is in the normal population. More precisely, Laczinsky in a 10 – year survey, found the prevalence of arthritis in psoriatic patients to be 6.8 percent, which is well above that of arthritis in the nonpsoriatic population.

Females show a greater tendency to develop psoriatic arthritis than do males.

Genetic evidence

Genetic factors are of demonstrable importance in the pathogenesis of psoriatic arthritis just as they are in psoriasis. Moreover, the findings favor a multifactorial form of inheritance, similar to that of psoriasis.

Among the environment influences, trauma is an important in precipitating psoriatic arthritis in genetically pre-disposed individuals. Thus, joint injury may be viewed as a Kobener's phenomenon similar to the Kobener's phenomenon – induced lesions of cutaneous psoriasis.

Clinical evidence

A more distinct clinical image of psoriatic arthritis has also emerged in recent years. It now appears clear that three major clinical types of psoriatic arthritis may be defined.

1. Patients with distal interphalangeal arthritis as the predominant clinical expression. This was the group of patients who for years were accepted as having “**Classic psoriatic arthritis**” since they clearly did not fit the usual pattern of digital involvement in rheumatoid arthritis which preferentially affects the proximal interphalangeal and metacarpophalangeal joints.
2. Patients with clinical arthritis mutants often complicated by digital “telescoping” and those showing involvement of the sacroiliac joints. Increasing attention has been paid to what has been called psoriatic spondylitis, which some regard as a very special variant of psoriatic arthritis. It is not yet clear whether psoriatic spondylitis is the equivalent of idiopathic ankylosing spondylitis.
3. Patients demonstrating an arthritic pattern indistinguishable from that of Rheumatoid Arthritis. These patients show the usual clinical elements of rheumatoid arthritis, such as early morning stiffness, symmetric involvement, fusiform swelling of the proximal finger joints ultimately ulnar deviation, rheumatoid nodules and commonly rheumatoid factor in the serum.

The onset of psoriatic arthritis is usually insidious. The age of onset is similar for psoriatic arthritis and rheumatoid arthritis. The traditional view that the onset of arthritis coincides with that of skin lesions is apparently no longer valid.

Actually a closed temporal relationship has been demonstrated for nail and joint involvement. Over 80 percent of patients with psoriatic arthritis have nail involvement, and a correlation between involvement of distal joints and the associated nails has been observed.

Other clinical findings that have been found at times in psoriatic arthritis include high fever, sclerodactyly, inguinal lymphadenopathy, absence of subcutaneous nodules, little muscle wasting, occasional tendon sheath effusions, ocular inflammation, and gastro – intestinal amyloidosis.

Radiologic evidence

X-ray examination of affected joints reveals a number of changes which are characteristic of psoriatic arthritis.

These include:

1. Predilection for distal interphalangeal joints with relative sparing of metacarpophalangeal, metatarsophalangeal, and proximal interphalangeal joints.
2. **"Whittling"** of terminal phalanges.
3. **"Pencil in cup"** changes.
4. Peripheral arthritis multians showing osteolysis and ankylosis.
5. Gross destruction of isolated small joints.
6. "Fluffy" periostitis.
7. Atypical spondylitis.

Laboratory evidence

The regular absence of Rheumatoid factor in the serum is the most important laboratory findings in psoriatic arthritis. Serologic tests for lupus erythematosus cells, antinuclear factor and other autoantibodies are also negative. The course of psoriatic arthritis can be expected to be chronic with remissions and exacerbations as in cutaneous psoriasis. Resolution of skin lesions may be followed by improvement of psoriatic arthritis but this is not constant relationship. Psoriatic arthritis tends to be less painful and disabling than rheumatoid arthritis. In severe cases, death is possible and not uncommonly results from complications of corticosteroid therapy.

Systemic associations

The major concomitant organ system involvement is psoriasis is inflammatory arthritis, acute anterior uveitis, inflammatory bowel disease and Reiter's disease. Certain patients with psoriatic arthritis may also show the rare complications of spondylitic heart disease, ocular inflammation, and gastrointestinal amyloidosis.

Diagnosis of Psoriasis

The above-mentioned histopathological findings are not specific for psoriasis alone.

There are no laboratory tests which will positively identify psoriasis. The blood count, urine analysis, ESR, and other haematologic, chemical and serologic studies are within normal limits in most cases of psoriasis.

Therefore the diagnosis of psoriasis is based upon:

1. The family history of psoriasis.
2. The typical distribution of the lesions on the scalp, elbows, knees, the front of the legs, back and nails.
3. Well-defined, non-indurated, dry, erythematous areas with silvery, layer-upon-layer scaling.
4. The candle – grease sign (when a psoriatic lesion is scratched with the point of a dissecting forceps, a candle-grease-like scale can be repeatedly produced even from the non-scaling lesions. This is CG sign)
5. Auspitz sign (Complete removal of a scale produces pin-point bleeding)
6. Koebner's phenomenon. (Psoriatic lesions may develop along the scratch lines in the active phase).
7. Little or no itching.
8. History of previous attacks, and seasonal variations of the disease.
9. Typical histopathology.

Differential diagnosis of psoriasis

In the majority of cases, the diagnosis of psoriasis is usually easy if the above mentioned features are borne in mind. Atypical cases may create diagnostic problems. The following conditions must be particularly considered in differential diagnosis.

(1) Seborrhoeic Dermatitis

The scalp patches are diffuse, ill-defined and moist; the hair is matted and tangled in the crust; the crusts are greasy. Body lesions affect the flexures, the sternal and inter-scapular regions. Sebo-psoriasis is a condition in which features both of psoriasis and seborrhoeic dermatitis are seen as indistinguishable.

(2) Syphilitic psoriasis

The history reveals an illicit exposure and the development of chancre; the rash is less scaly, and shows some degree of induration, mucous patches and lymphadenopathy. The V.D.R.L. is positive.

(3) Pityriasis rosea

A short history, centripetal distribution, a herald patch and typical oval lesions with cigarette – paper like, centrifugal scaling.

The flexural lesions must be distinguished from those in tinea cruris, intertrigo, seborrheic dermatitis; the nail lesions, from the lesions in tinea unguium, eczema, paronychia and syphilis; the palmar lesions, from the other causes of hyperkeratosis; the guttate variety, from lichen planus, and the erythroderma type from the other causes of erythroderma.

(4) Dandruff

The edges of psoriatic patches are sharp, dandruff patches are indefinite. The scale of psoriasis are dryer and more silvery than those of dandruff. The scales are moist and yellow.

(5) Ring worm: (Tinea circinata)

May resemble circinate psoriasis. But ring worm as a much greater tendency to show pustules or vesicles. The fungus can generally be found microscopically.

(6) Lichen Planus

A careful examination made in a good daylight will reveal the violaceous. The colour lichen planus as opposed to the red brown colour of

psoriasis. In lichen planus it is possible to find the characteristic polygonal, flat topped, shiny papules, on the front of the fore arm or wrist. Small white spots may be found inside the cheeks in about 50% of the cases. Lichen planus also itches more than psoriasis.

The flexural lesions must be distinguished from those in tinea cruris, intertrigo, seborrhoeic dermatitis; the nail lesions, from the lesions in tinea unguium, eczema, paronychia and syphilis; the palmar lesions, from the other causes of hyperkeratosis; the guttate variety, from lichen planus, and the erythroderma type from the other causes of erythroderma.

Prognosis

A permanent cure is not yet known, though individual attacks can, almost always, be controlled satisfactorily. Disease is non-infectious. General health and longevity are unaffected though the majority of patients suffer from the disease on and off throughout their lives. The course is chronic with varying periods of intermission (from weeks to years). The outlook is never either sure or bright, but one should avoid an attitude of defeatism. The whole position must be explained to the patient, and then he should be encouraged to persist with the treatment till all the lesions have disappeared; this brings down the relapse rate. The disease does not leave scars. There is only faint staining which disappears slowly. The nails gradually assume their normal appearance in months after the attack has abated.

Flexural, erythrodermic and pustular psoriasis take longer to heal than the typical variety. The palmar and nail lesions are rather resistant to treatment.

Complications

Complications in psoriasis are infrequent. The conditions which can complicate psoriasis are joint involvement (Psoriasis arthropathica) which can

caused by scratching and infection or the use of irritants; eczematization caused by scratching and infection or the use of irritants; lichenification brought on by scratching in neurotic individuals.

Treatment

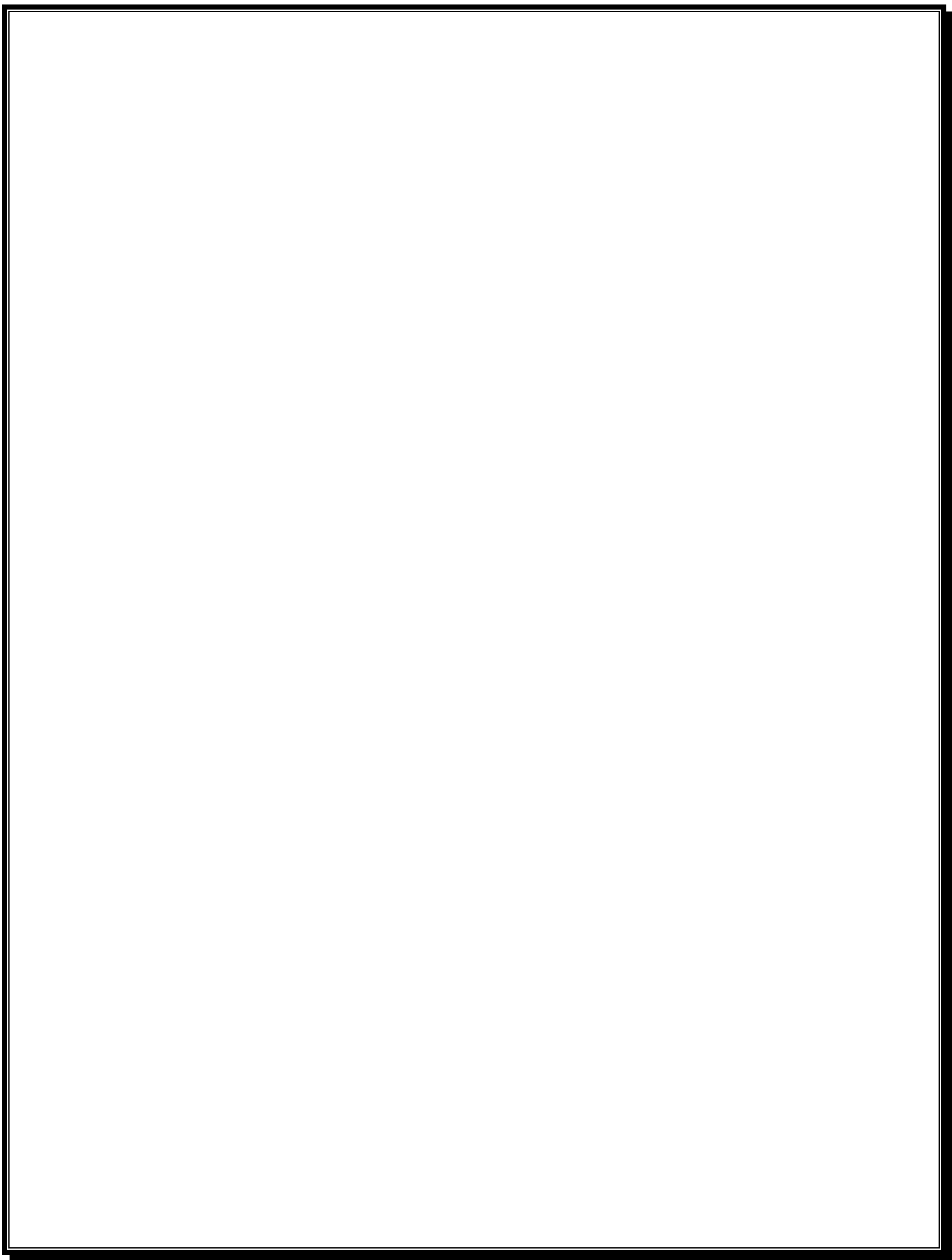
It is unsatisfactory in the sense that cure is out of question as the cause is unknown; hence the treatment is only palliative. The treatment should lay stress on :

MANAGEMENT

1. Impressing upon the patient that the treatment should be continued till the last lesion has disappeared. In this respect, the scalp should not be forgotten. The relapse rate is low, if the attack is completely controlled.
2. The general health of the patient should be maintained, and the exciting causes studied and eliminated, as far as possible. The patient's life should be regulated so that no undue stress affects either body or mind.
3. A moderate, warm climate, frequent sunbaths before the onset of the winter, and visits to sulphur springs, all of which are useful to bringing down the relapse rate. Natural sulphur baths should be taken during the holidays, especially in the winter.

DIET

The cutting down of fats, animal proteins and the quantity of food consumed.



MATERIALS AND METHODS

MATERIALS AND METHODS

Twenty cases in both male and female with various ages were selected. All the cases were clearly examined. Before admission for exact diagnosis of “**kalanchaga padai**” and to rule out any other co-existing systemic illness. All

other cases were found to have signs and symptoms of “**kalanchaga padai**”. These 20 cases were followed in In patient ward under the supervision of the Professor and Lecturer of the Post Graduate Sirappu Maruthuvam Department of Government Siddha Medical College, Palayamkottai. The above patients were discharged after a certain days under treatment and asked to attend the out patient department for further followup. Some of the patients those who are not willing for inpatients also treated in the out patients department and found very good prognosis.

Evaluation of clinical paremeters:

During interrogation of the cases were subjected to careful examination which invloves history taking and examination for clinical features. The peculiar sign and symptoms like dry well defined macules, papules and plaques of erythema with layer sign, koebrer’s phenomenos were taken into acoount.

A detailed history of the past history, allergee history, family history, personal history, dietary habits, residential places, occupational , socio-economicl status and prolonged ingestion of antimalrial drug for malarial fever, ulcer, a version, craving bladder and bowel habits were also taken before considering the case for selection in this study.

Clinical diagnosis of kalanchaga padai in siddha Aspect:

In siddha aspect the clinical diagnosis of “kalanchaga padai” is done by assessing the alteration in the following criteria.

1. Mukkutra nilai
2. Udal kattugal
3. Envagai thervugal

4. Paruvakaalam
5. Poriyal arithal
6. Pulanaal arithal
7. Vinathal

Besides these an individual case sheet was maintained for each case in the in-patient ward.

The clinical investigations:

Routine blood tests such as,

1. Total count
2. Differential count
3. Erythrocyte sedimentation Rate
4. Hemoglobin Estimation
5. Blood sugar,
6. Serum cholestrol
7. Blood urea.

Routine urine and stools examinations were done to rule out any systemic disease.

The pharmacological evaluation of the trial drugs and the bio-chemical analysis of the trial drugs were conducted respectively in the concerned department of the Government Siddha Medical College Hospital, Postgraduate Department of Sirappu Maruthuvam, Palayamkottai.

Management:

The mode of administration of the trial drugs are given below for both internal and external.

1. **Parangichakkai Churanam** : 1-3gms three times daily with water after meals.
2. **Thaasermeni Ennai**- applied externally over the affected areas.

Pathiyam or diet restrictions was strictly instructed to all the patients. **“Yoga practice”**, **“meditations”** and **“diet regimens”** were also advised to follow.

OBSERVATIONS AND RESULTS

OBSERVATION AND RESULTS

Interpretation of siddha parameter:

The inference obtained from the observation is explained as follows:

1. Family history,

2. Sex distribution,
3. Age distribution.
4. Religion distribution
5. Kaalam distribution
6. Occupational status.
7. Diet habit
8. Paruva Kaalam
9. Thinai reference
10. Socio economic status
11. Yakkai ilakkanam (physical constitution)
12. Gunam reference (Quality and characters)
13. Mode of onset
14. Duration of Illness
15. Clinical features
16. Distribution of three dosha
17. Udar kattukkal reference
18. Ennvagai thervugal
19. Neerkuri, Neikuri Experiment
20. Results after treatment

1. Family History:

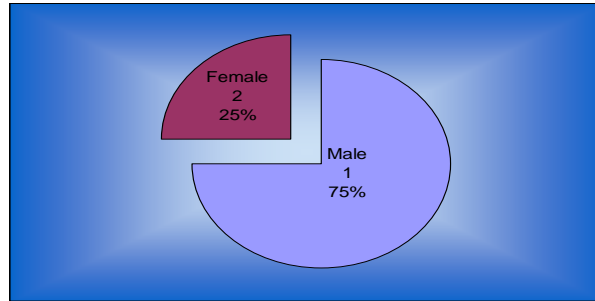
Though genetic is said to be the important cause, here 95% of patients showed negative family history.

2. Sex distribution

for the study of Kalanchaga padai, 20 patients were selected of which 75% cases were males and 25% of cases were females.

Sl no	Sex	No. of cases	Percentage
1.	Male	15	75%
2.	Female	5	25%

GRAPH SHOWING SEX DISTRIBUTION

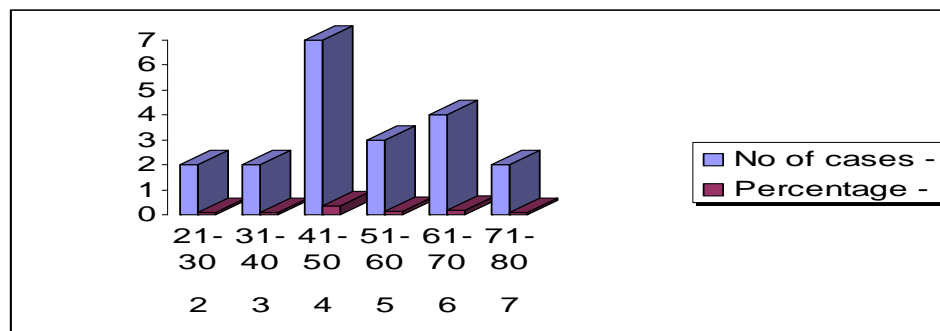


3. Age distribution

Out of the 20 cases taken for clinical trial, most of the cases were above 30 years of age.

Sl no	Age	No of cases	Percentage
1.	11-20	-	-
2.	21-30	2	10%
3.	31-40	2	10%
4	41-50	7	35%
5.	51-60	3	15%
6	61-70	4	20%
7	71-80	2	10%

GRAPH SHOWING AGE DISTRIBUTION



4.Kaalam

distribution

According to siddha literature human life can be classified into three periods, each of them having approximately 30 years age with respect to vatha, pitha and kapha dosha's dominance during different age period considering this into account.

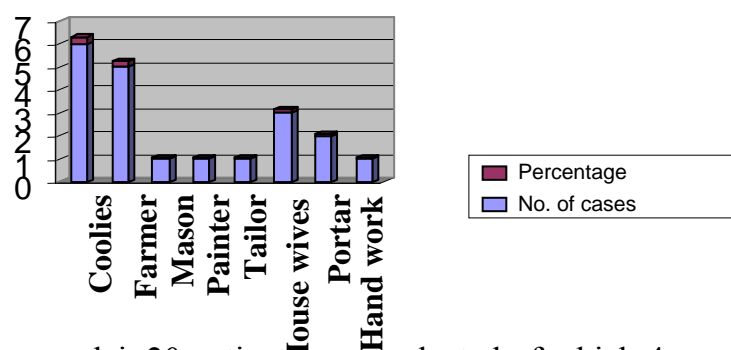
Sl.no	Kaalam	No.of cases	Percentage
1.	Vatha kaalam (1-33 Years)	2	10%
2.	Pitha kaalam (33-66 years)	16	80%
3.	Kapha Kaalam (66-100 years)	2	10%

5.Occupational Status

Out of 20 cases 25% of cases were farmers 30% of patients were coolies, 5% of case were masons, 5% of cases were painter, 5% patients were tailors, 15% of patients belong to house wives, 5% of cases were handworkers, and 10% were portars

Sl.no	Nature of work	No. of cases	Percentage
1.	Coolies	6	30%
2.	Farmer	5	25%
3.	Mason	1	5%
4.	Painter	1	5%
5.	Tailor	1	5%
6.	House Wives	3	15%
7.	Portar	2	10%
8.	Handwork	1	5%

GRAPH SHOWING OCCUPATIONAL DISTRIBUTION



6.Religion Distribution

For study on kalanchaga padai, 20 patients were selected of which 4 cases (70%) were Hindu religion case (5%) muslim religion and 5 cases [25%] were christian

Sl no	Religion	No. of cases	Percentage
1.	Hindus	14	70%
2.	Christian	5	25%
3.	Muslims	1	5%

7.Diet habits:

Sl.no	Diet habit	No..of cases	Percentage
1.	Vegetarian	4	20%
2.	Mixed	16	80%

Out of 20 cases, 80% were taking mixed diet and the rest 20% were vegetarian.

8.Paruva Kaalam:

Sl.no	Paruva kalam	No.of cases	Percentage
1.	Kaar kaalam (avani &Puratasi) Aug 16-Oct 15	1	5%
2.	Koothir Kaalam (Ippasi & Kaarthigai) Oct-16-Dec 15)	2	10%
3.	Munpani Kaalam Markazhi & thai Dec 16-Feb 15	11	55%
4.	Pinpani Kaalam (Maasi & panguni)Feb 16-Apr 15	4	20%
5.	Elavenil Kaalam (chithirai& Vaikasi)Apr 16-June 15	-	-
6.	Muthuveni Kaalam (Aani & Aadi) June 16-Aug 15	2	10%

Out of 20 patients, 5% cases were Kaar Kaalam, 10% were Koothir Kaalam, 55% were munpani, 20% were in pinpani and 10% were in muthuvenil kalam

9.Thinai reference

Sl.no	Thinai	No. of cases	Percentage
1.	Kurinji(Hill area)	-	-

2.	Mullai(Forest area)	-	-
3.	Marutham (Fertile Land)	18	90%
4.	Neithal (Coastal area)	2	10%
5.	Palai (Desert land)	-	-

Most of the patients (90%) in the clinical study belonged to the Marutha nilam, 10% of cases belonged to Neithal Nilam.

10.Socio economic status

Sl.no.	Socio Economic status	No. of cases	Percentage
1.	Poor	18	90%
2.	Middle	2	10%
3.	High Status	-	-

Out of 20 cases, 18 cases were from poor patients, 2 were from middle status.

11.Physical Constitution: (Yakkai ilakkanam)

Sl.No	Yakkai ilakkanam	No. of cases	Percentage
1.	Vatha Udal	-	-
2.	Pitha Udal	-	-
3.	Kapha Udal	-	-
4.	Thontha Udal	20	100%

All of the patients (100%) were belonged to Thontha udal.

12. Gunam (Quality and characters)

Sl.No	Gunam	No.of cases	Percentage
1.	Sathuva Gunam	-	-
2.	Rajo gunam	15	75%
3.	Thamo Gunam	5	25%

All of the patients (75%) had Roja gunam, 25% of patients had Thaamo gunam.

13.Mode of Onset:

Out of the 20 cases on clinical trial,90% of cases

Sl.no	Mode of onset	No. of cases	Percentage
1.	Acute state	2	10%
2.	Sub acute state	-	-
3.	Chronic State	18	90%

14.Duration of illness

Sl.No	Duration of months	No. of cases	Percentage
1.	1-2	10	50%
2.	3-4	5	25%
3.	5-6	1	5%
4.	7-8	-	-
5	8-12	1	5%
6.	13-24	1	5%
7.	25 and above	2	10%

15.Clinical features:

Sl.No	Clinical features	No.of cases	Percentage
1.	White patches with silvery scales	20	100%
2.	Scalp lesion	19	95%
3	Auspitz sign	20	100%
4	Koebner Phenomenon	13	65%
5.	Nail chanes	7	35%
6	Palm and sole lesion	1	5%

16.Distribution of thridosha

The derangement of doshas in kalanchaga padai is illustrated in the following table.

a) Table illustrating the derangement of vatha

Sl.No	Classification of vatha	No. of cases	Percentage
1.	Piranan	-	-
2.	Abanan	5	25%
3.	Udhanan	1	5%
4.	Samanan	20	100%
5	Vijanan	20	100%
6.	Nagan	-	-
7.	Koorman	1	5%
8.	Kirukaran	10	50%
9.	Devathathan	2	10%
10	Dananjayan	-	-

b) Table following the derangement of pitha

Sl.No	Classification of pitha	No.of cases	Percentage
-------	-------------------------	-------------	------------

1	Anar pitha	10	50%
2.	Ranjagam	15	75%
3.	Sathagam	20	100%
4.	Alosagam	-	-
5.	Prasagam	20	100%

Sathagam and prasagam were affected in all cases. Anar pitham was affected in 50% of cases. Ranjagam was affected in 75% of cases.

c) Table illustrating the derangement of Kapha.

Sl.no	Classification of kapha	No. of cases	Percentage
1.	Avalambagam	-	-
2.	Kilethagam	10	50%
3	Pothagam	-	-
4.	Tharpagam	1	5%
5.	Santhigam	3	15%

Only Kilethagam was affected in 50% of cases.

Tharpagam was affected in 5% of cases

Santhigam was affected in 15% of cases.

17.Udar kattugal

The seven thathus which contribute for the body structure and functions, get altered accordingly in pathological condition. Hence in kalanchaga padai, the following observations were tabulated as follows.

Sl.No	Udar kattugal	No. of cases	Percentage
1.	Saaram	20	100%
2.	Senneer	20	100%
3.	Oon	20	100%
4.	Kozhuppu	1	5%
5.	Enbu	-	-
6.	Moolai	-	-
7.	Sukkilam/Suronitham	-	-

18.Ennvagai Thervugal

In siddha system of science, the eight types of investigations were emphasized much for clinical approach and diagnosis of the malady. Hence the rules were strictly followed and the observations tabulated here.

Sl.No	Ennvagai thervugal	No.of cases	Percentage
1.	Naa	5	25%
2.	Niram	20	100%
3.	Mozhi	-	-
4.	Vizhi	5	25%
5.	Sparisam	20	100%
6.	Malam	5	25%
7	Moothiram	4	20%
8	Naadi a.vatha pitha b.Vatha kapha	13 7	65% 35%

19. Neerkuri, Neikuri reference:

Sl.No	Tyoe of test	No.of cases	Percentage
1.	Neerkuri “vaikkol niram”	20	100%
2.	Neikuri “muththothu nitral	20	100%

20.Results after treatment:

60% cases were good relief

20% cases were moderate relief.

20% cases were mild relief.

Sl. No	Results	No.of cases	Percentage
1.	Good relief	12	60%
2.	Moderate relief	4	20%
3.	Mild relief	4	20%

GRAPH SHOWING RESULTS

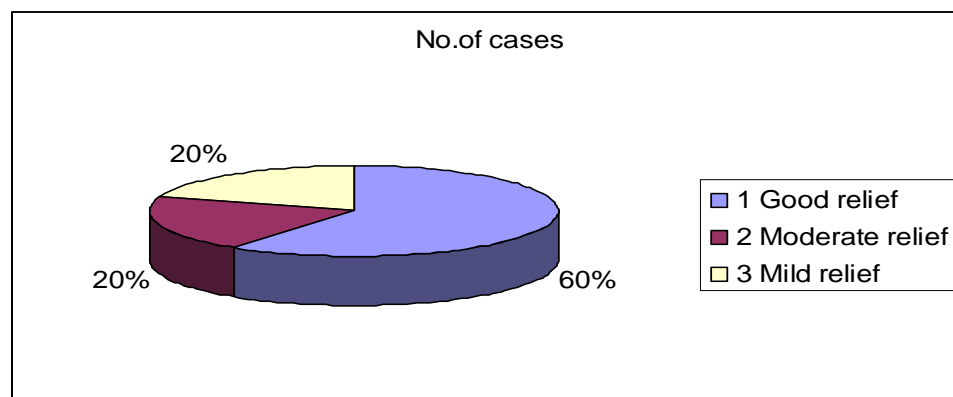


TABLE SHOWS BIOCHEMICAL REPORTS OF 20 IP CASES

S.NO	I.P.No	Name	Age/sex	Blood sugar mgs%	Blood urea mgs%	Serum Cholesterol mgs%	VDRL
1	529	Charles	44/M	113	19	157	NonReactive
2	668	Maharajan	50/M	99	20	192	NonReactive
3	1374	Gopal	75/M	75	23	203	NonReactive
4	1482	Ramaiah	65/M	85	21	178	NonReactive
5	2058	Lalitha	28/F	82	22	217	NonReactive
6	2677	Rayappan	54/M	89	23	173	NonReactive
7	2801	Sundarammal	45/F	103	32	165	NonReactive
8	56	Antoniammal	49/F	98	22	206	NonReactive
9	60	Sundaram	62/M	93	16	151	NonReactive
10	102	Kathiresan	40/M	374	32	160	NonReactive
11	138	Arumugam	60/M	142	22	154	NonReactive
12	139	Sudalai	75/M	100	40	154	NonReactive
13	190	Mary	45/F	108	21	250	NonReactive
14	207	Shunmugam	65/M	110	21	239	NonReactive
15	254	Shajahan	40/M	78	21	163	NonReactive
16	256	KalyanKumar	55/M	80	37	147	NonReactive
17	271	Mariammal	63/F	75	32	253	NonReactive
18	345	Periyasamy	42/M	197	23	142	NonReactive
19	276	Sarojini	25/F	84	21	165	NonReactive
20	615	Ramakrishnan	45/M	93	20	178	NonReactive

TABLE SHOWS RESULT 20 IP CASES

Sl.No	IP.No	Name	Age/Sex	Date of admission	Date of discharge	No. of days treated	Result
1	529	Charles	44/M	08.03.06	18.03.06	11 Days	Mild relief
2	668	Maharajan	50/M	23.03.06	26.05.06	65 Days	Good relief
3	1374	Gopal	75/M	15.06.06	10.07.06	26 Days	Good relief
4	1482	Ramaiah	65/M	28.06.06	07.07.06	11 Days	Mild relief
5	2058	Lalitha	28/M	06.09.06	20.09.06	15 Days	Moderate relief
6	2677	Rayappan	54/M	27.11.06	11.12.06	15 Days	Moderate relief
7	2801	Sundarammal	45/F	08.12.06	18.12.06	11 Days	Mild relief
8	56	Anotoniammal	49/F	09.01.07	24.01.07	16 Days	Moderate relief
9	60	Sundaram	62/M	09.01.07	29.01.07	21 Days	Good relief
10	102	Kathiresan	40/M	18.01.07	05.02.07	19 Days	Good relief
11	138	Arumugam	60/M	22.01.07	20.02.07	30 Days	Good relief
12	139	Sudalai	75/M	22.01.07	16.02.07	26 Days	Good relief
13	190	Mary	45/F	25.01.07	25.02.07	32 Days	Good relief
14	207	Shunmugam	65/M	25.01.07	15.02.07	22 Days	Good relief
15	254	Shajaghan	40/M	30.01.07	14.02.07	16 Days	Moderate relief
16	256	Kaliyankumar	55/M	30.01.07	23.02.07	25 Days	Good relief
17	271	Mariammal	63/F	30.01.07	17.02.07	19 Days	Good relief
18	276	Sarojini	25/F	31.01.07	25.02.07	20 Days	Good relief
19	345	Periyasamy	42/M	06.02.07	28.02.07	29 Days	Good relief
20	615	Ramakrishnan	45/M	26.02.07	09.03.07	12 Days	Mild relief

LIST OF OP PATIENTS

OP PATIENTS GIVEN ONLY INTERNAL MEDICINE

S.No	O.P No	Name	Age/Sex	Complaints			No of days treated	Result
				SL	S	P		
1	67608	Velayutham	75/M	+	+	+	36 days	Good
2	68340	Annathai	69/F	+	+	+	31 days	Good
3	68466	Meeran Mohideen	48/M	+	+	+	31 days	Good
4	71248	Allirani	40/F	+	+	+	20 days	Mild
5	2372	Jaanu Sharia	14/F	+	+	+	22 days	Mild

OP PATIENTS GIVEN ONLY EXTERNAL APPLICATION

Sno	O.P No	Name	Age/Sex	Complaints			No of days Treated	Result
				SL	S	P		
1	62918	Mookan	68/M	+	+	+	35 Days	Good
2	67233	Jeyanthi	35/F	+	+	+	31 Days	Good
3	68753	Sankarammal	47/F	+	+	+	31 days	Good
4	57705	Ramalakshmi	51/F	+	+	+	28 days	Moderate
5	69065	Azhagamma	45/F	+	+	+	30 days	Good

SL - Scalp Lesions

S - Scaling

P - Pinpoint Bleeding

OP PATIENTS GIVEN BOTH

INTERNAL MEDICINE AND EXTERNAL APPLICATION

S.no	O.P No	Name	Age/Sex	Complaints			No. of Days treated	Result
				SL	S	P		
1	38383	Gomathi	53/F	+	+	+	86 days	Good
2	63564	Chitra	23/F	+	+	+	31 days	Good
3	68546	Saraswathi	19/F	+	+	+	30 days	Good
4	69389	Nambi	50/M	+	+	+	36 days	Good
5	71162	Muthu	37/M	+	+	+	20 days	Good
6	74123	Avudaiammal	36/F	+	+	+	36 days	Good
7	74828	Jenitha	32/F	+	+	+	34 days	Good
8	74853	Sudalaimuthu	70/M	+	+	+	34 days	Good
9	76547	Amaravathy	37/F	+	+	+	28 days	Good
10	74612	Chandran	70/M	+	+	+	35 days	Good

SL - Scalp Lesions S - Scaling P - Pinpoint Bleeding

Name : Mary Age : 45 Sex : Female IP.No : 190

BEFORE TREATMENT



AFTER TREATMENT



Name : Kalyan Kumar Age : 55 Sex : Male IP.No : 256

BEFORE TREATMENT



AFTER TREATMENT



DISCUSSION

DISCUSSION

Kalanchaga Padai is a common skin disorder. It share special knowledge and skill in management. It is chronic disease that can be devastating for some people. Not just physically but emotionally. At times, the frustrating lack of available treatment can lead to a spiral of despair and hopelessness. Moreover, everyone wants to their physique so attractive, but this disease makes the skin silvery scaly appearance and the peel of skin is some what so apparent and obvious. The emotional needs of the patients should not be overlooked.

Perhaps, Kalanchaga Padai is not a contagious, disease. The affected individuals may be deprived from the society. Relapse and remissions of this disease is quite common and there may be no specific treatment in the other systems of medicine. But siddha system envisages the nature of the disease leading to the way of treatment is itself an achievement. In this way the present attempt is considered a mile stone.

INTERPRETATION OF SIDDHA PARAMETERS

For this dissertation more than 20 patients including both sexes were admitted in the in-patient ward of Post Graduate Department of Sirappu Maruthuvam, Govt.Siddha Medical College Hospital, Palayamkottai and they were given the trial drugs regularly.

They are interrogated thoroughly and their history, ailments , characters of signs and symptoms were noted in the materials and methods observation.

The interface obtained from the observation is explained as follows.

FAMILY HISTORY

Though genetic is said to be the important cause, here 95% of patients should negative family history.

CLINICAL FEATURES:

All the patients depicted the clinical features mentioned in the text book “**Siddha Maruthuvam Sirappu**”

SEX DISTRIBUTION:

Out of 20 cases 70% were males and 30% were females. So this disease can affect either sex.

AGE DISTRIBUTION :

During the course of the study the availability of the affected patients were in the age groups between 25-75 respectively.

KAALAM DISTRIBUTION:

The clinical study revealed the highest incident in Pitha kaalam (33-66 yrs) 80%.

OCCUPATION AND SOCIO ECONOMIC STATUS:

Out of 20 cases 90% were poor and 10% belongs to middle. The clinical study has Coolies, Farmers, Tailors, Hand workers, House wives, Painters and Mazon.

DIET HABIT:

Out of 20 cases, 55% cases were admitted during Munpani Kaalam, 5% were in Kaar Kaalam, 10% were in Koothir Kaalam, 20% were in Pinpani Kaalam and 10% were in Muthuvenil Kaalam. So this disease can occur in all Paruva Kaalam

THINAI REFERENCE :

Out of 20 cases, 90% were from Marutha Nilam and 10% were from Neithal Nilam. So this disease can be occur in all types of thinai distribution.

Basically Marutha Nilam is said to be a disease free land. But increasing the ratio is due to pollution , modernization of life style, stress and strain is now a days.

PHYSICAL CONSTITUTION :

All the selected cases were in the constitutional Thontha udal.

GUNAM: (Quality and Characters)

Out of 20 cases, 75% had Rajo gunam, 25% of patients had Thamo Gunam

From this inference one's character is very much important in developing disease. This was clearly stated on Siddha System of medicine. So control of mind and restriction of normal life style can be lead to reduction in the formation of disease.

MODE OF ONSET

90% of cases were in chronic state. Because of their ignorance and negligence of all the diseases, the patients did not come in the early stage.

DISTRIBUTION MUKKUTRAM

VADHAM:

In Kalanchaga Padai Abanan, Viyanan, Samanan, Kirukaran, Devathathan were affected.

Affected Abanan [30%] produce constipation

Affected Viganan [100%] produce Erythematous lesions in the affected skin.

Affected Samanan: 100% due to affection of their vayus.

Affected Kirukaran [45%] due to the loss of appetite and 5% excessive appetite

Affected Udanan [5%]-produce excessive thirst.

Affected Devathathan [10%]-produce excessive insomnia.

Affected Koorman [5%] – produce impairment in vision.

PITHAM:

AnarPitham, Prasagam, Ranjagam, Sathagam were affected.

Affected Anar Pitham [50%] Produce loss of affectite

Affected Ranjaga Pitam [100%] produce decreases Hb%, elevated ESR.

Affected Sathagam(100%) produce sluggishness.

Affected Pirasagam(100%) produce Discoloration , dryness and roughness of skin.

KAPHAM:

Kilethagam, Tharpagam and Santhigam were affected.

Affected Kilethagam [50%] produce loss of appertite.
Affected Tharpagam [5%]-produce burning sensation in the eyes .
Affected Santhigam [15%] produce painful joints.

UDAL KATTUGAL

In 7 udal kattugal Saaram, Senner, Enbu, Kozhuppu were affected.

Affected Saaram [100%] produce easy to fatiguability
Affected Senner(100%) produce erythematous lesions, reduced Hb concentration and elevated ESR.
Affected Oon (100%) produce weakness of sense organ.
Affected Enbu (15%) produce joint pain.
Affected Kozhuppu (5%) produce loss of activity.

Envagai Thervugal

Niram and Sparism were affected in all 20 cases.

Affected Sparism produce dryness of the skin , roughness, thickness and silvery scaly appearance of the skin.
Affected Niram produce greyish white discolouration of the affected skin
Malam was affected in 5 cases [25%] and constipation.
Naa was affected in 5 cases [25%] and produce Paled tongue .
Moothiram was affected in 4 cases [20%]. In that 5% produce was Polyuria and 15% were presence of Puscells in urine
Vizhi was affected 5 cases [25%] and produce paled conjunctives
Naadi: Vatha Pitha naadi in 13 cases vatha kabha naadi in 7

Regarding Moothiram, Neerkuri showed Straw coloured urine and the presence of puscells in urine in 3 cases and Albumin Trace in 2 cases and presence of sugar in 1 case. Neikuri showed **“Kabha Neer”**

Assessment of Result

Good relief	- significant disappearance of silvery scales.
Moderate relief	- Partial disappearance of lesions
Mild relief	- No obvious improvement in lesion.

60% of patients had good relief

20% of patients had moderate relief

20% of patients had mild relief.

The drugs under the clinical trial were,
Parangi Chakkai Churnam : 1-3 gms three times daily.
Thasermeni Ennai : for topical application

All the patients were strictly instructed to follow the **“Pathiyam”**

After their discharge, they were advised to take further Medicine through out patient Department, Sirappu Maruthuvam.

During their treatment and after discharge the patients were uniformly advised to follow Yogasanas and Pranayamam.

Laboratory investigation of Blood and urine were done once again after their completion of treatment. The increased ESR were noted to be normal.

The Diabetic Patients were advised to take further medicine for Diabetes mellitus.

SUMMARY

SUMMARY

The disease kalanchaga padai was taken for the clinical study. The clinical study on kalanchaga padai with reference to its aetiology, pathogenesis, investigation, clinical features, diagnosis and treatment were conducted at the post graduate department of sirappu maruthuvam, Govt. siddha Medical College, Palayamkottai. Twenty cases with the observation was made. They were gathered from the out patient ward and admitted in the in patient ward for the followings.

No drug reactions like nausea, vomiting, diarrhoea, flatulence, skin rashes, drowsiness were reported during the study period. The drugs employed in the clinical study were put to use only after careful purification process laid down for them individually.

The majority of the patients were male. The trial drug have mainly kaippu suvai and the mainly accounts for the therapeutic effect in skin diseases.

The results were found to be encouraging. Among the **in-patients** treated **60%** showed **good results**, **20%** showed **moderate results** and **20%** showed **mild results**. Along with the in patients, nearly 20 patients were treated under the out patient ward. **16 cases** showed **good results**, **2 cases** showed **moderate result** and **2 cases** showed **mild results**.

CONCLUSION

CONCLUSION

“Gtd ehafdpUe;Jk; nfsuth; Kd;
ghz;lth; gpd; Nghd njd;d tptunkdpd;
Neha;f; ftpo;j njd;gjd;wp kUe;jpw;F
tpidAKz;Nlh
ftiyAW Nehahsp Kd;dhAs; Ntjpapd;
gpd;fhl;rp NghNdha;
Jtu tbg;gJ Nghyf; fTut
Neha;fisf; nfhy;yj; njhlq;fpdhNd”
Njiuah; kUj;Jt ghujk;.

In **theraiyar Maruthuva bharatham**. The author defining, a physician must have a clear cut knowledge about the Causative factors , Normal physiological conditions, Pathological changes, Nature of its presentation and Progress of the disease before treating the patient

Investigations were found to be useful in assessing the progress of disease.

Siddha diagnosis was made on the basis of investigation like **Mukkuutra Nilaigal, Envagai thervugal, udal Kattugal, Kaalam**

A parallel modern diagnosis was arrived through routine Blood test , Urine tests, Stool examinations.

Treatment given for “Kalanchaga Padai” was on the basis of “Mukkuutra Theory”. The dearanged dhoshas were corrected by the medicine given.

gwq;fpr; rf;if #uzk; as an internal
and

jhrh;Nkdp vz;nza; as an external respectively .

The above medicines were selected from the siddha literatures .

rpfprrh uj;d jPgk;

and

Siddha Research Pharmacopeae respectively. Almost all the cases treated with above medicines were shown remarkable improvement .

No adverse effects were reported during the course of treatment and the drug evaluation was done with the modern parameters.

A length it is concluded that in the treatment of **Kalanchaga padai** with “gwq;fpr; rf;if #uzk; “ and jhrh;Nkdp vz;nza; were found to be very safe and also economical.

ANNEXURES

ANNEXURE-1

“PREPARATION OF TRIAL DRUGS”

cs; kUe;J : (Internal Medicine)

gwq;fpr; rf;if #uzk;

Njitahd ruf;FfSk;> msTfSk;:

gwq;fpr; rf;if - 280 fp

rptdhh; Ntk;G - 140 fp

rq;fd; Nth;g;gl;il - 70 fp

nts;sUF - 70 fp

rh;f;fiu - 350 fp



nra;Kiw:

xU kl;ghz;l;jpy; rJuf;fs;sp> jpUFfs;sp ,t;tpuz;ilAk; tiff;F vLj;J rpW Jz;lh;fp
ePh;tpl;L mjd; tha;f;F nky;ypa rPiyahy; NtLfl;b mjw;F Nky; gwq;fpr; rf;ifia rpW
Jz;lh;fp Ftpj;J Nky; rl;b Kb rPiy nra;J mLg;Ngw;wp MW kzp Neuk; vhpj;J rf;ifia
epoypWj;jp ,bj;J #uzpj;Jf; nfhs;s Ntz;Lk;. kw;w ruf;FfisAk; ntapypy; cyh;j;jp #uzk; nra;J
rh;f;fiuAk; Nrh;j;Jf; \$l;b GJ FLitapy; Nghl;L jhdpa Glk; gj;J ehl;fs; itj;J gj;jpug; gLj;jTk;.
msT: 1-3 fpuhk; tPjk; 2 –3 Ntis mDghdk; : ePh; my;yJ Njd;

jPUk; Neha;fs;:

fpue;jp> #iy> Nkfk;> ntl;il> gilfs;> tPuzk;> fz;lkhiy FzkhFk;.

gj;jpak;: fLF> vz;nza;> kPd;> khkprk;> Gif \$lhJ.

Mjhu Ehy;: “rpfr;rh uj;d jPgk;”

Mrphpah; : fz;Zr;rhkp (ak;)> gf;fk; 115.

“gwq;fpr; rf;if”

Botanical Name : Smilax China – Linn

NtW ngah;fs; : kJ];kpfk;> kJ];kpfp> rPd;gl;il> gwq;fpg;gl;il

Vernacular names:

English China root, Bamboo Briar root

Telugu: Pirangi-Chakka

Sanskrit:Madusnuhi

Hindi:Chobchini

Malayalam:China Pairu (or) pairu

Classification:

Divisions: Angiosperms

Class: Monocotyledons

Series : coronareal

Family:Liliaul

Genus: Smilax

Species: China.



Distribution

China, Japan, India, Himalaya mountain,

Organoleptic characters:

g.c. f;poq;F

Rit : ,dpg;G

jd;ik : jl;gk;

gphpT : ,dpg;G

g.c. fpoq;F

Therapeutic Actions:

1. cly; Njw;wp - Alterative
2. Nkfg;gpzptpyf;fp - Antisyphilitic
3. fhkg; ngUf;fp - Aphrodisiac
4. Jha;ikahf;fp - Depurative

Fzk;:

“jhfk; gythje; jhJel;lk; Gz;gpsit

Nkfq; fbfpu;jp tPo;%ye; - Njfkld;

Fl;il gfe;ju Nkw;nfh; tkdk; Nghk;

gl;ilapid Ar;rhpi;Jg; gh;

-Njiuah; Fzthfk;

ePh; Ntl;if> gw;gy tspNeha;> Gz;> gpsit> ePhpopT> fbtplk;> rpuq;F> %y%is> Klthjk;>
FiwNeha;> Iak;> gfe;jug;Gz;> the;jp ,it ePq;Fk;. Mz;ik cz;lhFk;.

Chemical constituents:

Root contains, Fat, Sugar, Glucoside, Saponin, Gum, Starch, Tannin, Resin and Cinchonin along with Smilacin and a Steroidal Sapanin yielding on hydrolysis.

Sarasapogenin have been identified in the tuberous roots. Flavionoid glycosides and three Saponins which yield Diosgenin also reported.

“rq;fd;” (Nth;g;gl;il)

Botanical Name : Azima Tetracanthu Linn

Nt.ng. rq;fQ;nrb> ew;rq;fd;> Kl;rq;fd;> rq;fpiyr;nrb

Vernacular names:

Tamil-Ichunka

Telugu -Tella-up

Hindi-kanta-gur-kamai

Bengali-Frikanta gati

Sanskrit-Kundovli

Malayalam-Changan

Marathi-Sorlikka port

Orriya-Odibhango

Kannada-Bileevuppi



Classification

Class-Dicotyledens

Subclass-Gamopetalae

Series-Bicarpellatae

Order-Gentianales

Family-Salvadoraceae

Genus- Azima

Species-Tetracantha

Distribution

It is cultivated throughout India.

Habit:

Bushy shrubs.

Organoleptic characters:

Rit ifg;G

jd;ik ntg;gk;

gphpT fhh;g;G

g.c. ,iy> ghy;. Nth;

Therapeutic actions:

rpWePh; ngUf;fp	- Diuretic
ntg;gKz;lhf;fp	Stimulant
Jth;g;gp	Astringent
cukhf;fp	Tonic
Kiwntg;gfw;wp	Anti-periodic
Nfhioafw;wp	Expectorant

Fzk,;

“rq;fk; Nth;g;gl;il rspapUkiyr; Ruj;ij mq;fthjf; fLg;ig Mlijg;igg; - gq;fNk
nra;Aq; fpue;jpiaAs; jPfhy; fpUkpiaapt;
itae; jdp nyhopf;F khy;”
- mfj;jpah; Fzthflk;

Chemical Constituents:

Leaves and stem contains 3 dimeric piperidine alkaloids. Namely,

- Azimine
- Azcarpine
- Carpaine

leaves contain

- Friedelin
- Glutinol
- Lupeol
- B -Sitosterd

“nts;sWF”

Bot.Name: Enicostemma Littorale Blume.

NtW ngah;fs;: mWF%yp> <athhpfk;> FNyhkp> rz;lk;> rf;fpu tPhpa ee;jd;> RNtjfp>
jpl;il> ntz;zW> rpNyl;Lkj;ij> gf;Ftk; gz;zp> fz;lhyp>

ghhf;;ftp.

vernacular names:

Tamil-vellari

Telugu-Neleguli, Nelagulimidi.

Mumbai-mamijwa

Hindi-Chootakirayta

Marathi-Kadavinaya, Mamijva, Tanavadu.

Sans-Krimihrita, Kisharakarma,

Kshitankshurpa, Mabhijaka, Magajika, Nahu,

Tiktapatra,

Bengal-Nagajirha

Ceylon-Vellarugu



Classification

Division: Angiosperms

Group:Dicotyledens

Class:Bicarpellate.

Series:Gentianales

Family: Gentianaceae.

Genus:Enicostemma.

Species-Littorale.

Distribution

The plant is distributed in all plains districts and 1,500 feet in the hills, often on cotton soil of India and Cyclone, tropical Africa, West Indies.

Habit: Glabrous perennial herb.

Part used: Whole plant.

Organoleptic characters:

Rit ifg;G

jd;ik ntg;gk;

gphpT fhh;g;G

Therapeutic actions:-

Stomachic - grpj;jPj;Jhz;b

Tonic - cly; cukhf;fp

Alterative - cly; Njw;wp

Laxative - kykpsf;fp

Febrifuge - Rukf;fp

Carminative - mfl;Ltha;tfw;wp

Anti-rheumatic

Nervine Tonic - euk;Gukhf;fp

Bitter Tonic

Fzk,:

“Fd;knkhL tha;T Fly;thjk; #iy apit

nrd;kk; tpl;Nlhbr; rpijAq;fhd; - td;Kiyaha;

cs;SUfp ue;jpnrhwp nahl;ba rpuq;FkWk;

nts;sWF jd;id tpUk;G”

jdhy; tspNeha;> fPy; gpbG;G> euk;Gfisg; gw;wpa foiy> jpdT> Fly;thA> Fd;kk;>
rpW rpuq;F Mfpait jPUk;.

Chemical constituents:

-Alkaloidal fraction

-Gentio crucine

-A monoterpene alkaloid-enicoflavin

-Glycoside

- Swertisioside apigenin
- Genkwanin
- Bitter glycoside Swertiamarin
- Isivitexin
- Swertisin
- Sapanarin
- Ophelic Acid

“rptdhh; Ntk;G”

Botanical Name: Indigofera aspalathoides Vahl.

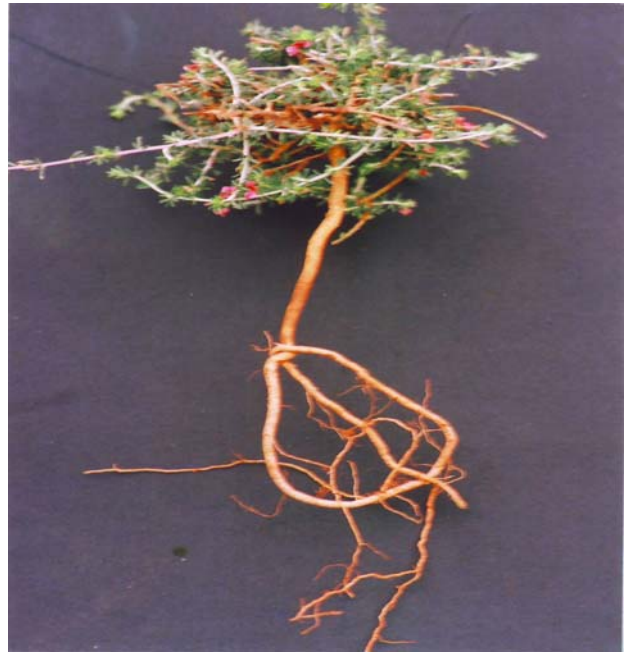
NtWngah;fs;: fhe;jhhp> mudpk;gk;> ,iwtd;> ,iwtd; epk;gk;> rpth> rptd;Nth;>
Kg;Gunkhpj;jhd; Nth;.

Vernacular names:

Sanskrit: Ratakohamba, Sivanimba
Tamil: Iraivan vemba, Sivanar vembu
Kannada: Nila, Sivamballi
Malayalam: Malali

Classification

Class: Dicotyledons
Subclass: Polypetalae
Series: Calycifloreae
Order: Rosales
Family Fabaceae
Genus: Indigofera



Species:Aspalathoides

Habit: Sub Shrub (rpW nrb)

Distribution: Commonly seen in South India, mostly growing on waste and barren grounds.

Organoleptic characters:

Rit ifg;G
jd;ik ntg;gk;
gphpT fhh;g;G
gad;gLk; cWg;G r%yk;

Therapeutic actions:

Emulant	cs;moy; Mw;wp
Stimulant	ntg;gk; cz;lhF;fp
Antiseptic	mOfyfw;wp
Disinfectant	fpUkp njhw;wfw;wp

Fzk;:

“Fl;lQ; rpuq;F Fiwg;Gg;Gr khe;ij
fl;lg; gpzpf; foYNk – jpl;lk;
cudpk;gq; fhaj;Jf; Fz;lhF Nkiy
mudpk;g nkd;D kUe;jhy;”
Njud; ntz;gh

.jdhy; Njhiyg; gw;wp tUk; gilfs;> ehl;gl;l Gz;> nrhwpfs;. gpsit> nfhLik nra;Ak;
tspNeha;fs;> FiwNeha;. ke;jk;> ghk;G eQ;R ePq;Fk;> moF cz;lhFk;.

Uses:

Leaves, flowers and tender root are demulcent, their decoction used in cancerous affections and leprosy also applied in abscesses.

Uses: „J miuj;J Ntisf;F xU nfhl;ilg;ghf;F msT gRtpd; ghypy; fyf;fp jpdKK; xU Ntis 3 ehs; rhg;gpl nrhwp> F\;lk;> gpuNkfk;> fpue;jp> Kjypaitfs; NghFk;.

“rh;f;fiu” (Saccharine Sugar)

“mUe;J kUe;jpw; fDghd khfg;

nghUe;Jkly; the;jp gpj;jk; Nghf;Fk; - tUe;jUrp

ePf;F kjpfgj;ij ePw;W kfpo;r;rpiaAz;

lhf;F eWQ;rUf;f iu”

nghUs;:

rh;f;fiuahdJ> kUe;JfSf;F mDghdkhf ,Ug;gJe; jtpu thjtkdj;ijAk;> gpj;j Njh\j;ijAk;> mNuhrfj;ijAk; Nghf;Fk;. nfl;bg;gl;l fgj;ij ,sf;fp kfpo;r;rpiaj; jUk;.

Therapeutic actions:

Antiseptic	- mOfyfw;wp
Demulcent	- cs;soyhw;wp
Tonic	- cly; cukhfp

ntsp kUe;J: (External Medicine)

“jhrh; Nkdp vz;nza;”

NrUk; ruf;FfSk;> msTfSk;:

Fg;igNkdp ,iy;rhW – 1800 ml.

Njq;fha; vz;nza; - 1800 ml.



fwpkQ;rs; nghb - 70 gm.

nra;Kiw:

Fg;igNkdp ,iyr;rhW kw;Wk; Njq;fha; vz;nza;> ,uz;ilAk; xU ghj;jpuj;jpypl;L
nfhjpf;f tplTk;. ijykhkJk; <ug;gjk; ed;F Rz;b nkOFgjk; tUk;NghJ kQ;rs; nghbiar; Nrh;j;J
ed;F fyf;fp tpl Ntz;Lk;. gpd; 1 my;yJ 2 nfhj te;jTld; ghj;jpuj;ij mLg;gpy; ,Ue;J ,wf;fTk;.
gpd; ijy;ij tb fl;b Nrfhpj;Jf; nfhs;sTk;.

cgNahfk;: ntspg; gpuNahfk;

msT : Njitahd msT

jPUk; Neha;fs; : gilfs;> fpue;jp> gTj;jpuk;> fug;ghd;> jPUk;

Mjhuk; : rpj;jh hprh;r; gh;NkhNfhgpah

Mrphpah; : Dr.G.D.ehAL kw;Wk; Dr. M.rz;KfNtY>L.I.M., H.P.I.M>

“Fg;igNkdp”

NtW ngah;: mhpKQ;rh> G+idtzq;fp> Nkdp> khh;[hyNkhfpdp> Fg;ghkzp>
mhpl;lKQ;rh>

Vernacular names:

Sanskrit –Aritta manjarie

English- Indian Acalypha

Hindi- Kupp, Khokali

Bengali-Muktajhuri

Gujarat-Vanchi Ranto

Marati- Khoki, Khajoti, Kuppiyuta, Muripindi



Tamil: Kuppaiveni, kuppaimeni

Kannada:kuppigida

Malayalam:kuppamani

Kongani:Kunkniphall

Oriya:Indiramaris

Sinhalese:Kupa-menya

Classification

According to Bentham and Hookers classification(1867) *Acalypha Indica* Linn(Kuppaimeni)classified under:

Class:Dicotyledone

Subclass:Monochlamydeae

Series: Unisexualis

Family: Euphorbiaceae

Genus: *Acalypha*

Species:Indica

gad;gLk; cWg;G: Parts Used.

Leaves, Root, Stacks and Flowers

,iy> Nth;> rk;%yk;

Distribution:

Acalypha indica linn is distributed throughout the tropical and subtropical region. It is seen in all plain districts and in the lower hills at south India.

Identifying Character:

Habit: An erect annular herb, attaining a height of 30-100 cms, branches numerous, long, ascending, angular, finely pubescent

Leaves: simple-ovate or thromboid ovate, 2-5-7.5 cms long with slender petioles.

Flowers:

Unisexual.

Parts used: leaves, root, stalks, and flowers.

Chemical constituents of *acalypha indica* in the plant contains a Cyanogenic, glucosides and two alkaloids *Acalyphin* and *Triacetonamine*.The other constituents are

n-octocosanol, B-Sitosterol, Tannin, Resin and Essential oil.

Therapeutic Action of Acalypha Indica.

mfl;Ltha;tfw;wp-Carminative

kykpsf;fp-laxative

GOf;nfhy;yp -anthelmintic

rpWePh;g;ngUf;fp-diuretic

Nfhio mfw;wp-Expectorant

the;jp cz;lhf;fp-Emetic

#jfk; cz;lhf;fp-Emmenagogue

nghJFzk;:

je;j %yg;gpzp jPj;jpL Gz; rh;ttplk;

te;J Fd;kk; thjk; cju%ye;jpdT

RyQ;R thrk; njhlh;gPe rha;fgk; Nghk;

Qhyq;nfhs; Nkdpajdh;"

-Njiuah; Fzthflk;

Fg;igNkdp ,iyahy; gy;yb Neha;> jPr;Rl;l Gz;> gaph;tif eQ;R> tapw;F typ> tspNeha;>

%yk;> eikr;ry;> Fj;jy;> ,iug;G> %f;F ePh; gha;jy;> Nfhio Mfpait jPUk;

“kQ;rs;

Ntw;Wg; ngah;;: mhprdk;> eprp, gPjk;> fhe;jhhpg;gif

Turmeric

Vernacular names:

English-Turmeric



Hindi-Ben,Haldi

Arab-Kunkum

Telugu-Pasupu

Kannada-Arishina

Marathi:Gujarathi-Haldi, Halada

Bengali-Haldi, Pitras

Malayalam-Mannal, Marinalu

Sanskrit-Haridra, Harita, Lakshmi, Mangalya, Pavithra, Shiva, Shobana, Suvarna,
Uma, Yoshitapriya and Durga.

Classification:

Class :Monocotyledons

Subclass: monocotyledone

Series:Epigynae

Family-Scitamineae

Sub-family-Zingiberace

Genus-curcuma

Species-Longa

Parts used: Rhizome

Native of the plant: Southern Asia, India

Description :Perennial herb, horizontal tuberous rhizomes

Chemical Constituents

-Curmcunoids

-Choleteric acid

-Vanillic acid

- Turmerone

- Zedoamdiol

-Zingiberine

- Curcumin
- Terpene
- Pinene
- Hepato 1-4-6-triene -31
- Hepato 1-6-diene
- Curlone
- Curzerenone
- Cymene
- Fatty Acids
- Alpha Atlantone

Analysis India Turmeric(Rhizome)

Moisture 13.1%

Protein 6.3%

Fat 5.1%

Mineral Matter 3.5%

Fibre 2.6%

Carbohydrate 6.9%

(Carotene as Vit.A) 50IU/100mg.

The essential Oil (5.8%)

Distillation of dry Rhizomes :-

 d- Pellandrene 1%

 d- Sabinene 0.6%

Lincol 0.5%

Zingiberene 25%

Sesquiterpene 58%

(Turmerol)

Organoleptic Characters:

Rit - ifg;G> fhh;g;G

jd;ik - ntg;gk;

gphpT - fhh;g;G

nra;if:

cly; cukhf;fp Tonic

Jaulf;fp Anodyne

tPf;fq;fiur;rp Deobstruent

mfl;LthAtfw;wp Carminative

grpj;jP Jhz;b Stomachic

mOfyfw;wp Antiseptic

“jiytyp eP Nuw;wQ; risahj Nkf

KiyTjU gPerj;jp DhNl – typRug;G

tpQ;R fbtP\Kk; tPw tpuzq;fSk;Ngh

kQ;rl; fpoq;Ff;F khy;”

gjhj;j Fz rpe;jhkzp

jiytyp> ryNjh\k;> gpuNkfk;> ehrpfhNuhfk;> tPf;fk;

ngUtpuzk; ,itfs; jPUk;.

“ Njq;fha; vz;nza; “ [oil of cocus nucifera]

[Coconut oil]

Chemical Constituents:

Free caprylic acid,

Glycorides of Lauric, Myristic, Palmitic and Stearic acid.

Therapeutic actions:

cs;soyhw;wp - Demulcent

cly;Njw;wp - Alterative

Fzk;-

“Njq;fhap nda;ajdhw; wPahy; tLGz;Nghk;
ghq;fhf; \$e;jy; glh;e;NjW – ePq;fhj
gy;ybapd; NdhAk; glh;jh kiu rpuq;F
ky;ywg; Nghnkd; wwp”

-“gjhh;j;j Fz rpe;jhkzp”

Njq;fha; vz;nzahy; jPg;Gz;> gy;ybNeha;> glh;jhkiu> rpuq;F ,itfs; NghFk;. \$e;jy;
tsUk;.

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ANNEXURE II

GOVT. SIDDHA MEDICAL COLLEGE, PALAYAMKOTTAI BIO- CHEMICAL ANALYSIS OF PARANGICHAKKAI CHOORANAM

Preparation of the extract:

5gms of chooranam was weighed accurately and placed in a 250 ml clean beaker. Then 50 ml distilled water is added and dissolved well. Then it is boiled well for about 10 minutes. I t was cooled and filtered in a 100 ml volumetric flask and then it is made up to 100 ml with distilled water. This fluid is taken for analysis.

Qualitative analysis:

S.NO	Experiment	Observation	Inference
1.	<u>TEST FOR CALCIUM</u> 2ml of the above Prepared extract is taken in a clean test tube . Add 2 ml of 4% Ammonium oxalate solution is added to it	A white precepsitate is formed	Indicate the presence of calcium

2.	<u>TEST FOR SULPHATE</u> 2ml of the extract is added to 5% barium chloride solution	A white preceipitate is formed	Indicates the presence of sulphate
3.	<u>TEST FOR CARBONATE</u> The extract is treated with silver nitrates solution	No white preceipitate is formed	Absence of chloride
4.	<u>TEST FOR CARBONATE</u> The substance is treated with concentrated HCL.	No brisk effervescence is formed	Absence of carbonate
5.	<u>TEST FOR STARCH</u> The extract is added with weak iodine solution.	No blue colour is formed	Absence of starch
6.	<u>TEST FOR IRON FERRIC:</u> The extract is treated with glacial acid and potassium Ferro cyanide	No blue colour is formed	Absence of ferric Iron
7.	<u>TEST OF IRON FESRROUS.</u> The extract is treated with concentrated Nitric acid and ammonium thio cynate	Blood red colour is formed	Indicates the presence of Ferrpus Iron
8.	<u>TEST FOR PHOSPHATE</u> The extract is treated with ammonium Molybdate and concentrated nitric acid	No yellow preceipitate is formed	Absence of phosphate
9.	<u>TEST FOR ALBUMIN</u> The extract is treated with ferric chloride	No yellow preceipitate is formed	Absence of albumin
10.	<u>TEST FOR TANNIC ACID</u> The extract is treated with Esbach's reagent	Blue black preceipitate is formed	Indicates the presence of Tannic acid
11.	<u>TEST FOR UNSATURATION</u> potassium permanganate solution is added To the extract	It gets decolourised	Indicates the presence of unsaturated compound
12.	<u>TEST FOR THE REDUCING SUGAR</u> 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2mts.	Colour change occurs	Indicates the presence of Reducing sugar.

13.	TEST FOR AMINO ACID One or two drops of the extract is placed on a filter paper and dried it well after drying 1% Ninhydrin is sprayed over the same and Dried it well	violet colour is formed	Indicates the presence of Amino acid.
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ANNEXURE III PHARMACOLOGICAL ANALYSIS

ANTI – HISTAMINE EFFECT OF PARANGICHAKKAI CHOORANAM

AIM :

To study the anti-histamine effect of parangichakkai chooranam by Bio-Assay – Method .

Preparation of the test drug :-

1. gm of parangichaikkam Chooranam was suspended 10 ml of water . This was used for the evaluation and pharmacological studies. 1 ml of suspension contains 100 mg of test drug.

SOLUTION REQUIRED :

Histamine (1 in 1, 00, 000 strength)

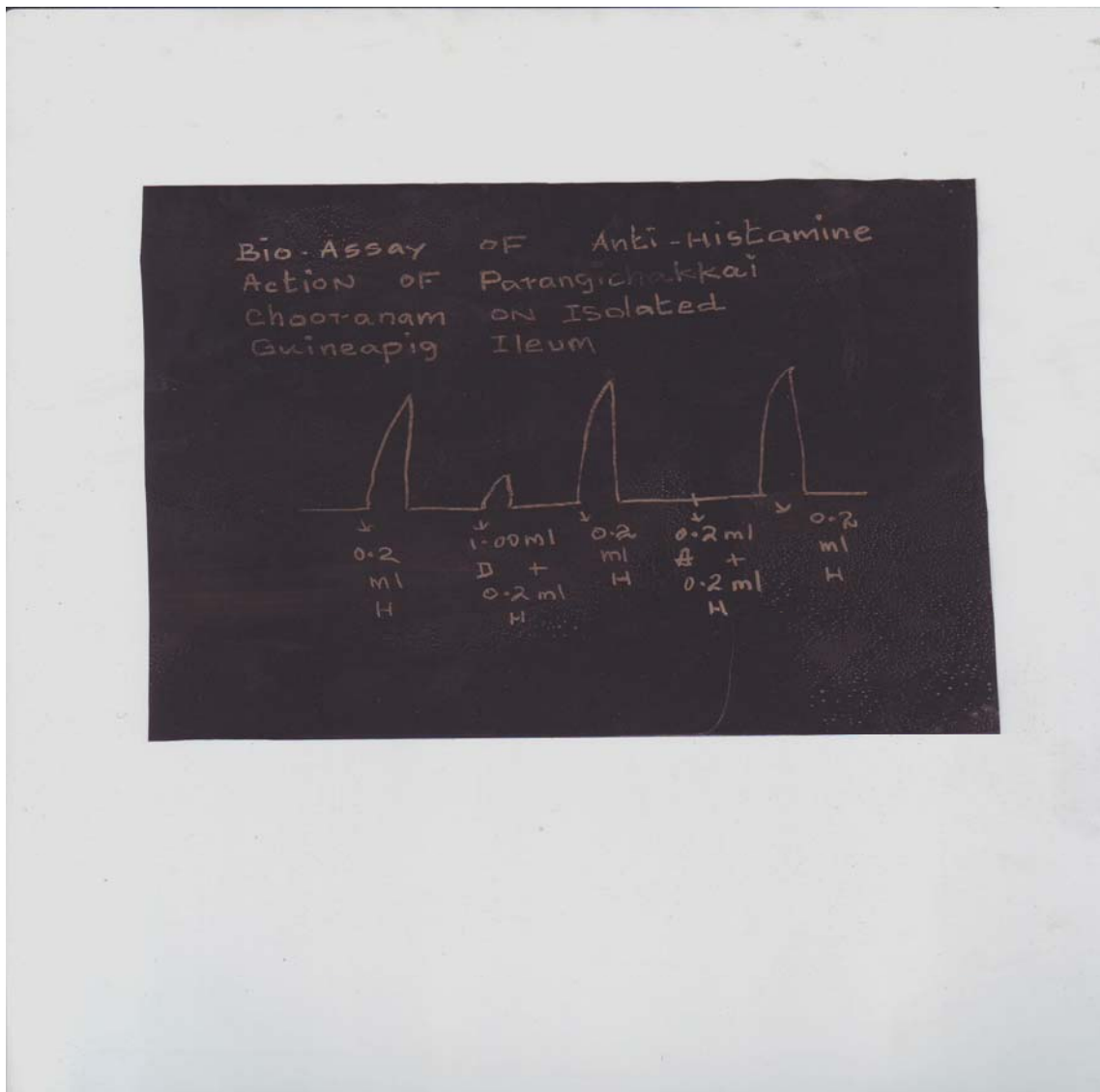
METHOD

A guinea pig weighing about 450 gms was starved for 48 hours and only water was allowed to drink . It was killed by stunning with a sharp blow on the head and cutting its throat to bleed it to death . The abdomen was immediately opened and the viscera inspected and loops of intestine identified ,using the patch as a landmark . Then the ileum was removed and placed in a shallow –dish containing the warm “Tyrode solution “. With the help of 25 ml pipette , the lumen of the length ,generally 4 cm , in a fully relaxed state and the sutures were made with needle and tied at either ends . The segment is suspended in an isolated organ bath.It was aerated by an oxygen tube and immersed in Tyrode solution at 37°C Drugs were given to study the inhibitory effect of histamine induced contractions .

INFERENCE

The drug has significant anti-histamine action.

ANTI – HISTAMINE EFFECT OF PARANGICHAKKAI CHOORANAM



ANALGESIC STUDY OF PARANGICHAKKAI CHOORANAM

AIM:

To study the analgesic effect of Parangichakkai Chooranam on Albino rats by tail flick method .

PREPARATION OF THE TEST DRUG :

1 gm of parangichakkai chooranam was suspended in 10 ml of hot water as suspending agent .1 ml contained 100mg of the test drug .

EQUIPMENT: Hot water bath

PROCEDURE :

Six male albino rats (weighing -80-100 gms)were used in three groups .The animals were allowed to free access to food and water until they brought for the experiment .The animals which showed the positive response to the stimulus within a given time were selected for the study.

After the selection of animals which were responding to stimulus within 2 seconds , they were divided into 3 groups , each group consisting of 2 rats .

The hot water was maintained at 55°C . The tip of the tail was immersed into the water bath and the time was noted when the rat flicked the tail.

First group was given the Parangichakkai Choornam at a dose of 100 mg/100 gm body weight of the animal.

Second group was administered with Paracetamol at a dose of 20 mg/100 gm of body weight. Third group was given to the 1 ml of water and kept as control.

After the drug administration, the reaction time of each rate after ½ an hour, 1hour and 1½ hour were noted in each group. (When a rat fails to flick the tail, it should not be continued beyond 8 seconds to avoid injury) and the average was calculated.

The results of control group, standard group and drug treated group were tabulated and compared.

Sl.no	Group	Dose/100gm body weight	Initial reading in Seconds	After ½ hour	After 1hr	After 1½ hr
1	Control	Water 1 ml	2.5	2.5	2.5	3.0
2	Standard	Paracetamol 20 ml / 1 ml	2.5	4.0	5.0	6.5
3	Test Drug	Parangichakkai Chooranam 100mg / 1 ml	2.5	3.5	4.5	5.5

Inference

From the above tabulation it is noted that Parangichakkai Chooranam has **Significant** Analgesic action.

ACUTE ANTI INFLAMMATORY ACTION OF PARANGICHAKKAI CHOORANAM

Aim

To demonstrate the acute anti-inflammatory activity of Parangichakkai Chooranam in Albino rats by Hind Paw Method.

Preparation of the test drug:

1 gm of Parangichakkai Chooranam was suspended in 10 ml of water. From the above test drug 2 ml was administered orally. 1 ml contain 100 mg of Parangichakkai Chooranam.

Procedure:

Six Albino rat weighing 100-150 gm were taken and divided into three groups and each group consisting 2 rats.

First group was kept as control and received water. Second group received Ibu brufen at a dose of 20 mg/100gm body weight. Third group of animals received Parangichakkai Chooranam.

Before administration of drugs, the hind paw volume of all rats was measured. This was done by dipping the hind paw up to the tibio tarsal junction in a mercury Plethysmograph. Soon after the measurement the drug was administered. One hour after the administration of drug a subcutaneous injection of 0.1ml of 1% w/v of carrageenin in water was made into plantar surface of both the hind paw of each rat.

Three hours after carrageenin infection, the hind paw volume was measured once again. Difference between the initial and final value were noted and compared.

The method is more suitable for studying anti inflammatory activity on actual inflammation.

The result of the drug is compared with the standard as well as control group.

S.No	Group	Dose/100 gm body weight	Initial Reading average	Final reading average	Mean Difference	Percentage of Inflammation	Percentage of Inhibition
1	Test	Parangichakkai Chooranam 100 mg / 1 ml	1.0	1.45	0.45	50	50
2	Standard	Ibu brufen- 20mg/1ml	0.80	0.85	0.05	6.25	93.75
3	Control	Water-1ml	0.65	1.5	0.85	100.0	-

Inference:

From the above experiment it is observed that the test drug Parangichakkai Chooranam has Moderate acute anti inflammatory action.

CHRONIC ANTI INFLAMMATORY EFFECT OF PARANGICHAKKAI CHOORANAM

Aim:-

To study the chronic anti-inflammatory activity of the test drug parangichakkai chooranam by cotton pellet method.

Preparation of the test drug:-

1gm of parangichakkai chooranam was suspended in 10ml of water. From the above test drug 1ml was administered orally. 1ml contain 100mgm of parangichakkai chooranam.

Procedure:-

Cotton-pellets each weighing 10mg were prepared and sterilized in an autoclave for about one hour under 151bs atmospheric pressure. 6 rats weighing between 100-200gms were selected and were divided into 3 groups , each containing 2 rats. Each rat was anaesthetized with ether and cotton-pellets were implanted, subcutaneously in the groin two in each side.

From the day of implantation one group of animal received parangichakkai chooranam in a dose 100mg/100gm of body weight other groups of animals were received distilled water 1ml/100gm of body weight.

Next group received Ibu brufen in a dose of 20mg/100gm of body weight.

On the eighth day the rats were sacrificed and the pellets were removed and weighed. Then dried in an incubator at 60°C to 80°C and then it weighed.

The weight of the granulation tissue formed is the difference between net weight and dry weight. The results of the control and test group were compared and the results are tabulated.

Effect Of Parangichakkai Chooranam:-

S.no	Group	Dose/100mg body weight	Mean weight of granulation tissue	Percentage of Inflammation	Percentage of inhibition
1.	Control	Distilled water 1ml	250mg	100.0	-----
2.	Test drug	Parangichakkai chooranam 100mg/1ml	133mg	53.0	47.0
3.	Standard	Ibu brufen 20mg/1ml	56mg	22.4	77.6

Inference

The drug shows moderate chronic anti-inflammatory action.

**ACUTE ANTI INFLAMMATORY ACTIVITY OF THAASERMENI ENNAI
(EXTERNAL USE)****Aim :**

To study the acute Anti-inflammatory activity of the test drug Thaasermeni Ennai by hind-Paw method in Albino Rats.

Procedure:

Six healthy Albino rats weighting 100-150 gm were taken and divided into three groups, each consisting at 2 rats.

First group was kept as control by giving water of 2 ml/100gm of body weight. The second group received Ibu brufen at a dose of 20mg/100gm of body weight. The third group was kept as test group.

Before application of test drug, the hind-paw volume of all rats were measured. This was done by dipping the hind-paw (up to tibio-tarsal junction) into a mercury Plethysmography. While dipping the hind-paw, by pulling the syringe piston, the level of mercury in the centre small tube was made to coincide with red marking and reading was noted from the Plethysmograph.

On hour later, a subcutaneous injection of 0.1ml of 1%(W/V) carrageenin in water was made into planter surface of both hind-paw of each rat. To the test group, Thaasermeni Ennai was topically applied for three times over the inflamed surface in a thin layer within 30mts gap. To the other groups no drug was applied over the inflamed surface.

One and half an hour after injection the hind – paw volume was measured once again . The difference between the initial and final volume would show the amount of inflammation . Taking the volume in the control groups as 100% of inflammation and the anti-inflammatory effect of the test group is calculated

Effect of Thaasermeni Ennai:-

S.no	Group	Dose/100gm Body weight	Initial reading average	Final reading average	Mean difference	Percentage of Inflammation	Percentage of inhibition
1	Test drug	Thaasermeni Ennai/Ext	0.9	1.55	0.65	72.2	27.8
2	Standard	Ibu brufen- 20mg/ml	0.80	0.85	0.05	6.25	93.75
3	Control	Water-1ml	0.65	1.5	0.85	100.0	-

Inference:-

It is observed that Thaasermeni Ennai has **mild acute anti-inflammatory action** .

**ANTI-HISTAMINE EFFECT TO THAASERMENI ENNAI
(EXTERNAL APPLICATION)**

Aim:

To study the anti-histamine effect of Thaasermeni Ennai by Bio-Assay method.

Preparation of the test drug :

1 ml of Thaasermeni Ennai was used for the studies.

Solution required:

Histamine (1 in 1,00,000 strength)

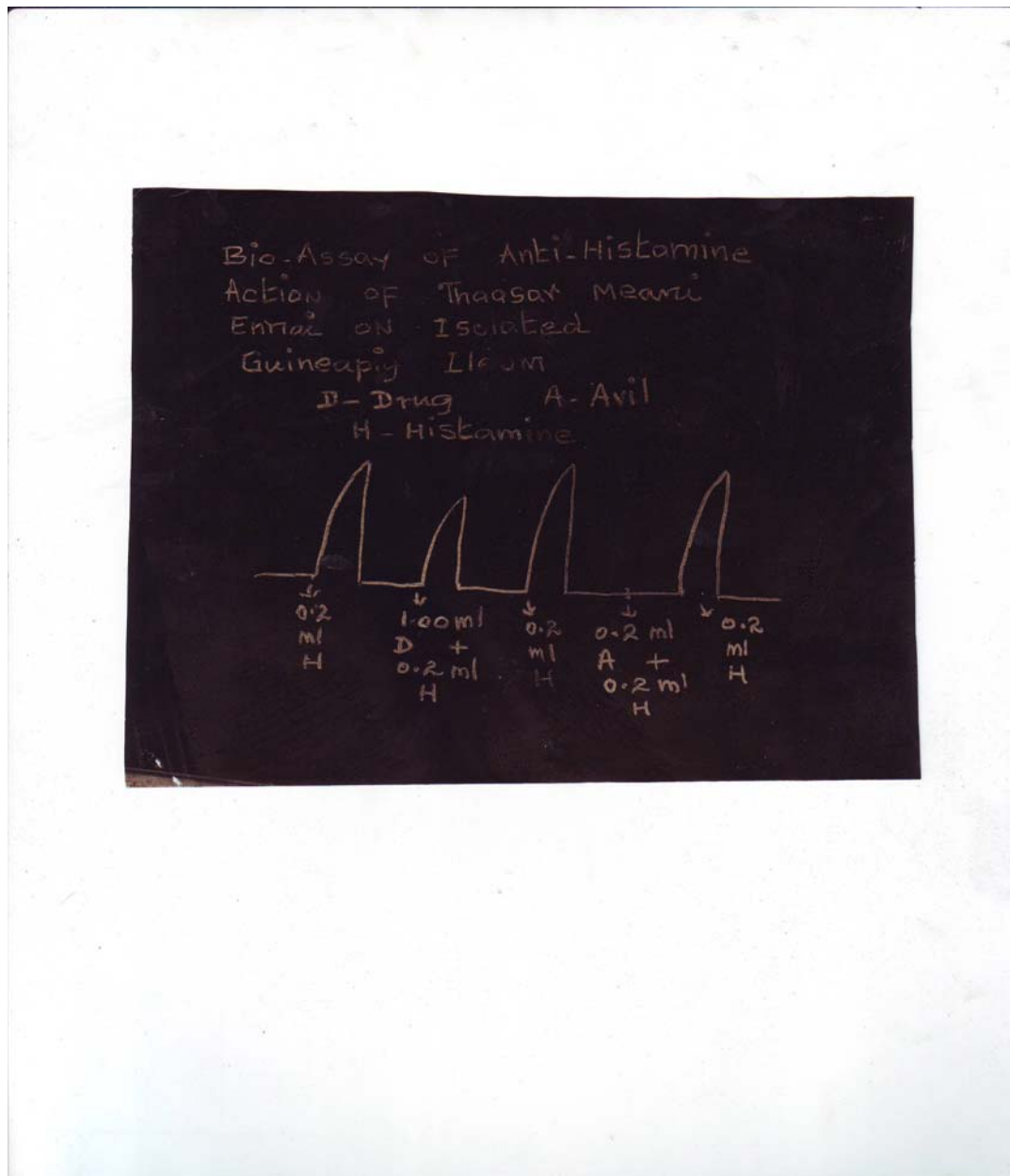
Method:

A guinea pig weighting about 450 gms was starved for 48 hours and only water was allowed. It was killed by stunning with a sharp blow on the head and cutting its throat to bleed it to death. The abdomen was immediately opened and the viscera inspected and loops of intestine identified using the patch as a land mark. Then the ileum was removed and placed in a shallow-dish containing warm "Tyrode solution". With the help of 25 ml pipette, the lumen of the length, generally 4 cm, in a fully relaxed state and the sutures were made with needle and tied at either ends. The segment is suspended in an isolated organ bath. It was aerated by an oxygen tube and immersed in Tyrode solution at 37° C. Drugs were given to study the inhibitory effect of histamine induced contractions.

Inference:

The drug has moderate **Anti-histamine actions**.

ANTI-HISTAMINE EFFECT OF THAASERMENI ENNAI



ANNEXURE IV

GOVERNMENT SIDDHA MEDICAL COLLEGE AND HOSPITAL
POST GRADUATE SIRAPPU MARUTHUVAM, PALAYAMKOTTAI

TIRUNELVELI-627002

CASE SHEET PROFORMA FOR “KALACHAGA PADAI”

I.P No	:	Occupation	:
Bed No	:	Income	:
Ward	:	Nationality	:
Name	:	Religion	:
Age	:	Date of Admission	:
Sex	:	Date of Discharge	:
Permanent	:	Diagnosis	:
Address	:		
Temporary	:	Result	:
Address	:	Medical Officer	:

Complaints and duration :

History of Present Illness :

History of Previous Illness :

Personal History including

 Habits :

Allergic History :

Family History :

GENERAL CONDITIONS OF EXAMINATION

1. Consciousness :

2. General appearance :

3. Stature :

4. Nourishment :
5. Skin Changes :
6. Facies :
7. Pallor :
8. Jaundice :
9. Cyanosis :
10. Clubbing :
11. Lymphadenopathy :
12. Abdominal distension :
13. Jugular venus pulsation :
14. Engorged Veins :
15. Koilonychia :
16. Paedal oedema :
17. Generalised oedema :
18. Temperature :
19. Pulse :
 - Rate : /mm
 - Rhythm :
 - Volume :
 - Character :
 - Peripheral pulses :
20. Respiration
 - Rate :
 - Rhythm :
 - Character :
21. Heart
 - Rate : /mm
22. Blood Pressure

CLINICAL EXAMINATION-SKIN

Inspection:

Site (area) :

Size :
Shape :
General colour of the skin :
Colour of the lesion :
Scalling :
Oedema :
Vesicles :
Thickend of Lichenified :
Papules :
Exdation :
Macules :
Erythema :
Inflammatory/Non-inflammatory :

PALPATION:

Sensation :
Nature of Lesion :

rpj;jKiw Njh;T

I. epyk;

FwpQ;rp
Ky;iy
kUjk;
nea;jy;
ghiy

II. gUtfhyk;

fhh;fhyk;(Mtzp-Gul;lhrp) -
\$jph;fhyk;(Ig;grp – fhh;j;jpif) -
Kd;gdp (khh;fop – ij) -
gpd;gdp (khrp – gq;Fdp) -
,sNtdpy; (rpj;jpiu-itfhrp) -
KJNtdpy; (Mdp-Mb) -

III. ahf;if (cly;)

thjk; -
gpj;jk; -
fgk; -
fyg;G -

IV. Fzk;

rj;Jt Fzk;
,uNrh Fzk;
jNkh Fzk;

V. nghwpGyd;fs;

nka; (njhLczh;T) -
tha; (Rit) -
fz; (ghh;it) -
%f;F (ehw;wk;) -
nrtp (Nfl;ly;) -

VI. fd;Nke;jphpak;/fd;kltplak;

if (jhdk;) -
fhy; (fkdk;) -
tha; (trdk;) -
vUtha;(tprh;f;fk;) -
fUtha;(Mde;jk;) -

VII. cl;fha mjf;fhak;

Gak; (Forearm)	-
rak; (Arm)	-
fhy; (Leg)	-
ghjk; (Feet)	-

VIII Kk;kyk;

kyk;	-
%j;jpuk;	-
tpah;it	-

IX. gpw cWg;Gfspd; epiy

,Ujak;	-	
Gg;Grk;		-
,iug;ig	-	
fy;yPuy;		-
kz;zPuy;		-
rpWFly;		-
ngUq;Fly;		-
rpWePufk;		-
rpWePh;g;ig		-
%is		-
fUg;ig	-	

X. caph; jhJf;fs;

(m) thjk;

gpuhzd;		-
mghdd;		-
tpahdd;		-
cjhdd;	-	
rkhdd;	-	
ehfd;		-
\$h;kd;	-	
fpUfud;		-
Njtjj;jd;		-
jdQ;nrاد;		-

(M) gpj;jk;

mdw; gpj;jk;		-
,uQ;rf gpj;jk;	-	
rhjf gpj;jk;		-
MNyhrf gpj;jk;		-
gpuhrf gpj;jk;		-

(.) fgk;

mtyk;gfk;	-	
fpNyjfk;	-	
Nghjfk;	-	
jw;gfk;	-	
re;jpfk;	-	
XI cly; jhJf;fs;		
rhuk;	-	
nre;ePh;		
Cz;	-	
nfhOg;G	-	
vd;G	-	
%ois		
Rf;fpyk;/RNuhzpjK;		
XII vz; tifj; Njh;Tfs;		
ehb	-	
];ghprk;	-	
eh	-	
epwk;	-	
nkhop	-	
tpop	-	
kyk;		
epwk;	-	
vil	-	
,Wfy;	-	
,sfy;	-	
%j;jpuk;		
m. ePh;f;Fwp		M. nea;f;Fwp
epwk;	-	
kzk;	-	
vil	-	
Eiu	-	
vQ;ry;		

MODERN ASPECTS

Examination of other Systems:

1. Respiratory System.
 - a. Shape of the Chest :
 - b. Position of the Trachea :
 - c. Type of breathing :
 - d. Added sounds (if any) :
2. Cardio Vascular System :
3. Gastro Intestinal System :
4. Central Nervous System :
5. Excretory System :

LABORATORY INVESTIGATIONS

1. Blood:

TC:	Cells/ cu mm		
DC:	P: % ; L: %;	E: %;	
ESR:			
	½ hour	:	mm
	1 hour	:	mm
Hb%		:	
Blood Sugar		:	
Serum Cholesterol		:	
VDRL		:	

2. Urine

Albumin	:
---------	---

Sugar :
Deposits :

3. Motion

OVA :
Cyst :
Rbs :
Pus Cells :

- 4. Skin Scrapping /Clipping :**
5. Culture and Sensitivity :
6. Others :

VERUPADUTHI KANTHAL (Differential Diagnosis)

THEERUM THEERA NILAI (Prognosis)

MARUTHUVA MURAI

Progress Note	
Date	Progress of the patient

**GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL
POST GRADUATE SIRAPPU MARUTHUVAM,
PALAYAMKOTTAI TIRUNELVELI-627002.**

I.P.No	:	Occupation	:
Bed No	:	Income	:
Ward	:	Nationality	:
Name	:	Religion	:
Age	:	Date of Admission	:
Sex	:	Date of Discharge	:
Permanent Address:		Diagnosis	:
Temporary Address:		Result	:
		Medical Officer	:

Clinical Pictures

Sl.No	During Admission	During Discharge

--	--	--

Place:

Date:

Regarding of Progress

SL.No	Clinical Features (Signs & Symptoms)	1 week	II weeks	III weeks	IV Weeks
1.	Itching				
2.	Macules				
3.	Oedema				
4.	Border				
5.	Vesicles				
6.	Oozing				
7.	Pustules				
8.	Scaling				
9.	Lichenification				
10.	Varicose Vein				
11.	Erythema				
12.	Ulcers				

Theerum Theera Nilai :

Line of Treatment :

11 Days	Mild relief
15 Days	Moderate relief
15 Days	Moderate relief
11 Days	Mild relief
16 Days	Moderate relief
21 Days	Good relief
19 Days	Good relief
30 Days	Good relief
26 Days	Good relief
32 Days	Good relief
22 Days	Good relief
16 Days	Moderate relief
25 Days	Good relief
19 Days	Good relief
20 Days	Good relief
29 Days	Good relief
12 Days	Mild relief

Nil	NAD	Nil	Nil	Nil	Nil	- Ve
Nil	NAD	Nil	Nil	Nil	Nil	- Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
+	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve
Nil	NAD	Nil	Nil	Nil	Nil	-Ve

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--	--	--	--	--	--	--

ent count, P-Polymorph, L-Lymphocytics, E-Eosinophils, Hb-Haemoglobin,
 -Before Treatment, AT-After Treatment.

ings %				ings %		
BT	AT	BT	AT	BT	AT	
113		19		157		NoReactive
99		20		192		NoReactive
75		23		203		NoReactive
85		21		178		NoReactive
82		22		217		NoReactive
89		23		173		NoReactive
103		32		165		NoReactive
98		22		206		NoReactive
93		16		151		NoReactive
374		32		160		NoReactive
142		22		154		NoReactive
100		40		154		NoReactive
108		21		250		NoReactive
110		21		239		NoReactive
78		21		163		NoReactive
80		37		147		NoReactive
75		32		253		NoReactive
197		23		142		NoReactive
84		21		165		NoReactive
93		20		178		NoReactive

5/M	+	+	+	Good	36 days
0/F	+	+	+	Good	31 days
3/M	+	+	+	Good	31 days
0/F	+	+	+	Mild	20 days
4/F	+	+	+	Mild	22 days

Only External Application

	Complaints			Result	No of days Treated
	SL	S	P		
	+	+	+	Good	35 Days
	+	+	+	Good	31 Days
	+	+	+	Good	31 days
	+	+	+	Moderate	28 days
	+	+	+	Good	30 days

+	+	+	30 days	Good
+	+	+	36 days	Good
+	+	+	20 days	Good
+	+	+	36 days	Good
+	+	+	34 days	Good
+	+	+	34 days	Good
+	+	+	28 days	Good
+	+	+	35 days	Good